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AIR-RAID INJURIES OF THE CHEST.[1]

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PEOPLE with chest injuries form a very small percentage of the total air-raid casualties reaching the surgeon's hands, the reason being that most chest wounds are severe and cause death.

In many manuals of surgery, it is often categorically stated that chest injuries are purely in the province of the chest specialist, and that operations should not be performed without a specialist anæsthetist accustomed to endotracheal anæsthesia; and it is advised that all chest casualties should be immediately transferred to a "chest centre". This is, of course, the ideal course; but in actual fact it cannot always be achieved, owing to such circumstances as the impossibility of transport over bombed roads. The hospital of which I was in charge in London was in the centre of a heavily bombed area, and though casualties could be received, it was often impossible to transfer patients for some days. We had to deal with all wounded, including chest casualties, and anyone treating people who have sustained air-raid injuries must be prepared to deal with any type of wound in any part of the body. Most wounds of the chest are as urgent as compound fractures and need just as prompt attention.

Chest injuries in air raids may be due to the bomb itself, machine gun bullets from low-flying aircraft, or to shrapnel from anti-aircraft shells falling to the ground. The effects of an exploding bomb may be divided into three subsections: (*a*) Shrapnel wounds from the burst bomb casing. (*b*) Direct effects of the blast or suction wave: (i) a bomb bursting in close proximity, leading to dismemberment or death; (ii) a bomb bursting further away, causing injury by hurling the patient to the ground or against some solid object; (iii) lung blast itself. (*c*) Injuries due to the effect of the bomb explosion on buildings: (i) crushing injuries of the chest, of varying grade of severity; (ii) injuries due to flying missiles, varying from blocks of masonry to bricks, stone or glass.

Examining these causes, one may state that the type of lung injury sustained will depend largely on the type of surroundings in which the patient finds himself when the air raid occurs. If he is in a building, shelter or cellar, he may sustain lung blast from a bomb exploding in the room in which he is sheltering. The house may be wrecked and fall on him and cause severe crushing injuries of the chest. If the patient is caught in a street, he may suffer lung blast if the bomb falls anything up to 150 yards away from him, and if he is in the direct line of the blast. He may suffer crushing or even perforating lesions from flying bricks, masonry or glass. In a town unprotected by a balloon barrage, he may be hit by machine gun bullets, deliberately aimed at him from low-flying enemy aircraft, or he may be hit by falling pieces of shrapnel from the anti-aircraft barrage. In the open country, the source of danger lies in the bullets of the enemy machine and in the blast of exploding bombs.

Classification of Chest Injuries.

If injuries of the heart and mediastinum are omitted, there are two main types of chest injuries: (i) non-penetrating wounds, (ii) penetrating wounds. It is necessary to remember that these casualties may receive multiple injuries of the chest and other systems, including

[1] Read at a meeting of the North-Eastern Medical Association on August 2, 1941, at Lismore, New South Wales.

abdomino-thoracic wounds. For example, a patient of mine was standing in the street when a bomb fell. He was struck anteriorly by a piece of stone, which severed the right internal mammary artery and then entered the pleural cavity. At the same time, the force of the explosion hurled him to the ground, fracturing some ribs posteriorly. The fractured ribs were driven into the lung, severely lacerating it.

Non-penetrating injuries vary according to the force causing them. A force, widely diffused, such as a bomb explosion, will cause the condition of lung blast. Here the blast causes the chest wall suddenly to smack the lung, which is then affected by areas of hæmorrhage throughout the lung field, most prominent in the sub-pleural region. Lung blast varies in severity from those injuries causing death or serious illness to those causing slight chest pain and cough. The effects of lung blast appear usually twenty-four to forty-eight hours after injury, when dulness to percussion, tubular breath sounds and crepitations in the lung field will be found.

A less diffuse force may cause severe injury to the lungs without severe injury to the chest wall; there may be large rents or tears in the lungs, and the lung may even be torn off its hilum (so-called subcutaneous injuries of the lung). The intercostal vessels may be torn, with resulting hæmothorax. Bleeding from lung vessels may occur; but this is usually stopped by compression exerted on the lung by a coincidental pneumothorax or the hæmothorax itself, owing to the low pressure in the pulmonary vessels. This does not apply to hæmorrhage from the bronchial arteries, in which the blood pressure is systemic and three times that of the pulmonary vessels.

Crushing injuries will cause fractures of ribs, which may be multiple, and so segments of the thoracic wall may be unsupported, severe distress and shock being caused. At the same time, fractured ribs may cause severe lung injury and lacerations.

These last two types of injuries will be complicated by hæmothorax or pneumothorax or by a combination of both. In certain types of lung injury in which the lungs are torn, air may be able to enter the pleural cavity but not leave it, owing to the opening's being valvular in nature; this will give rise to a "tension pneumothorax". These cases are usually easy to diagnose; bruising of the chest wall, obvious signs of fractured ribs and surgical emphysema are present. There may be hæmoptysis, though it is surprising what extensive lung damage may be present without this, owing possibly to a contraction of the muscles of the bronchioles leading to the injured portion of the lung.

The signs of hæmothorax are too well known to need repetition. A tension pneumothorax can be diagnosed by the increasing cyanosis and dyspnœa of the patient, the absence of breath sounds, the tympanitic percussion note and the shift of the apex beat away from the affected hemithorax. A tension pneumothorax unaccompanied by a penetrating wound of the thoracic wall means lung injury which need not necessarily be severe. It may be associated with a rapidly spreading surgical emphysema.

A patient with a movable thoracic wall due to many fractured ribs—usually fractured anteriorly and posteriorly—suffers from severe pain, shock and dyspnœa. Many of these patients have injury to the intercostal nerves causing reflex abdominal guarding, which must be differentiated from that caused by intraabdominal lesions.

Penetrating injuries vary from the wounds caused by a bullet or a piece of glass to large wounds caused by pieces of shrapnel or brick. A bullet may cause a small wound if it penetrates between the ribs, but if it hits a rib this may be smashed and severe internal wounds be caused, the seriousness being masked by the small wound of entry. These objects usually take clothes and dirt with them.

In these penetrating wounds there are the following types: (*a*) penetrating wounds with retained foreign body; (*b*) traversing wounds with entrance and exit wounds; (*c*) tangential wounds, in which the object strikes a rib and then goes through the diaphragm into the abdomen, possibly causing no lung injury; (*d*) abdomino-thoracic wounds. All these wounds will be complicated by hæmothorax or pneumothorax of varying degree. The diagnosis of these injuries is usually self-evident from an inspection of the wound; but the deeper injury may require further investigation.

Treatment.

There are certain types of lung injury which require immediate attention, either at the scene of the "incident" or at the first-aid post.

Non-penetrating injuries, consisting of multiple rib fractures resulting in a "flexible chest wall", cause severe shock and dyspnœa, and are treated by immediate strapping from below upwards in the usual routine manner. The strapping must cross well over the mid-line, both in front and behind.

Tension pneumothorax is treated by the insertion of a large bore serum needle through an intercostal space—for example, the second intercostal space medial to the mid-clavicular line. This procedure may have to be repeated, or the needle kept *in situ*.

Penetrating wounds must be cleaned and an aseptic dressing applied until the patient reaches an operation centre.

In cases of open pneumothorax, especially the so-called "sucking wound", the following measures must be carried out immediately. Obvious superficial foreign bodies should be removed and visible bleeding parts must be ligated. Intercostal hæmorrhage is stopped by the tying of a ligature round the ribs themselves, both behind and in front of the bleeding point. The pneumothorax opening must be closed by large pads and strapping as a first-aid measure. Morphine should be given in adequate doses.

We may now follow the patients to a centre where the chest casualties can be completely treated. These centres must not be too far away, as such casualties stand transportation badly. First, they need to be operated on within six to eight hours. Secondly, the patient must be treated for shock with morphine, the intravenous administration of plasma or blood transfusion and the application of warmth; oxygen, up to 90% concentration, may be necessary. Antitetanic serum and gas-gangrene antiserum are given. These patients are treated in a semi-sitting position by means of a bed rest, once the shock is overcome. As soon as the patient's general condition is satisfactory a more complete examination must be made. A portable X-ray machine should be on hand so that chest radiographs can be taken to aid in diagnosis and in the localization of foreign bodies. This may also disclose the unsuspected presence of contralateral pulmonary collapse or of lung blast.

Certain chest wounds require priority over others, namely: (i) "sucking wounds"; (ii) severe internal hæmorrhage, as shown by rapidly increasing hæmothorax, rising pulse rate and increasing pallor; (iii) wounds of the lower part of the chest, which are usually abdomino-thoracic in character.

I shall now discuss the detailed treatment in hospital of patients with chest wounds.

Non-Penetrating Injuries of the Chest.

In non-penetrating injuries of the chest the treatment aims at the stopping of further blood loss, the elimination of positive pressure in the pleural cavity, and the elimination of excessive mobility of the thoracic wall due to fractured ribs.

A patient with an ordinary closed pneumothorax requires no more than conservative treatment, such as rest in bed.

The first-aid treatment of tension pneumothorax has already been discussed. If this is continuous, a needle may be left in place and attached to a rubber tube draining into an antiseptic solution. If the tension pneumothorax is severe and persistent, it is due to a ruptured bronchus, and exploratory thoracotomy must be performed.

Hæmothorax may be due to small lacerations of the lung, which quickly stop hæmorrhaging, or to bleeding from intercostal vessels, large lung vessels or hilar vessels. When effusion into the pleura has been considerable there

may be associated signs of loss of blood. The effusion is increased by fluid secreted from the irritated pleura itself.

Unless hæmorrhage is continuing, as shown by a rising pulse with a falling blood pressure and increasing pallor, conservative treatment is used—rest in bed, the exhibition of morphine and careful observation. Some surgeons immediately put air into the pleural cavity with an artificial pneumothorax machine in order to raise the intrapleural pressure and so stop any further hæmorrhage.

Till recently it was taught that these patients should be left alone; but it is now realized that many patients with a hæmothorax, when the blood is left *in situ*, later develop a thick fibrous parietal pleura, which is very severe in its effects and leads to a fixed hemithorax and lowered vital capacity. Accordingly, at the end of a period varying from forty-eight to seventy-two hours, the blood should be aspirated. This blood is usually fluid, owing to a lack of tissue juices to inhibit heparin, which stops the blood from clotting. If, however, there is much lung injury, clotting will occur from the liberation of tissue juices and the blood cannot be aspirated. Therefore rib resection should be performed, the blood clot removed and drainage instituted. This will stop the later ill-effects and will also help to prevent an infected hæmothorax. Should hæmorrhage continue, however, then the chest must be explored.

Surgical emphysema may occur from fractured ribs, or may accompany a tesion pneumothorax, in which case it spreads rapidly. It may also arise in the mediastinum from ruptured bronchi, spread through the upper aperture of the thorax and cause respiratory embarrassment. In these cases the insertion of a hypodermic needle into the subcutaneous tissues and massage of the air to the needle will be all that is necessary.

With regard to lung blast, all persons near an exploding bomb must be suspected of suffering from this condition and treated prophylactically with rest in bed in hospital. Lung blast may not appear clinically for twelve to twenty-four hours; but an early X-ray examination will reveal it before the clinical signs appear. The treatment of the condition is symptomatic. The important point is early rest in bed.

Penetrating Wounds of the Chest.

The majority of patients with thoracic wounds will need operation; simple through-and-through wounds from rifle bullets or small pieces of shrapnel may be treated expectantly, however, though these may require treatment for complications. All other patients should be submitted to operation as soon as the general condition permits this, especially if evidence of internal bleeding is present. The sooner the excision of the wound is performed, the less the chance of later infection. After twenty-four hours there is, of course, no need for surgical measures to prevent infection—it is too late. One must watch the patient, and if the temperature rises wide opening of the chest and drainage will be necessary.

The general principles of operative treatment are the following. All wounds of the chest wall are radically excised, both soft tissues and fractured bones. They may be dusted with sulphanilamide powder. If the pleura is found to be open, even if there is only a small hole, the wound is extended in an intercostal space, or if the wound does not give good access, a new incision is made in the sixth or seventh intercostal space postero-laterally, according to the position of the original wound. All blood is removed from the pleural cavity by suction or swabbing. Foreign bodies are removed from the lung, care being taken that fragments of clothing as well as pieces of metal are looked for. Any lacerated lung tissue is excised and the wounds are sutured with catgut. A severely injured lobe may need lobectomy. The chest wall is carefully sutured. The pleural space is drained, to remove any effusion or prevent the development of a tension pneumothorax should a broncho-pleural fistula occur.

Pre-Operative Treatment.—Such measures of resuscitation as plasma infusion and the exhibition of sulphapyridine as a prophylactic measure must be considered.

Anæsthetic.—In general some form of positive pressure apparatus, such as the gas anæsthetic machines at present in use, should be employed in order to steady the mediastinum during coughing when the chest is wide open. This apparatus can be used to deliver nitrous oxide and oxygen by a tight-fitting face-piece.

Intratracheal anæsthesia is not necessary—in fact it may lead to a severe post-operative tracheitis.

"Pentothal Sodium" used intravenously may be given as a basal narcotic to be followed by a general anæsthetic. Local anæsthesia is not advisable in war wounds, owing to the likelihood of late severe infection.

The Technique of Exploratory Thoracotomy.—In exploratory thoracotomy the entrance wound, and when one is present, the exit wound, must be first excised. The excision may include the removal from the track of spicules of broken ribs or scapula. In many cases one may use the original wound to enter the thoracic cavity. It may have to be enlarged and the chest entered through the affected intercostal space. In other cases, in addition to excision of the wounds a deliberate exploration of the chest must be made through a fresh incision, as in penetrating wounds in the posterior triangle of the neck. The skin incision is made over the seventh rib by an oblique postero-lateral approach. The superficial and deep muscles are divided and the ribs and intercostal spaces are exposed. When the upper part of the chest is more obviously involved, an opening is made through the sixth intercostal space, and when the lower part is involved through the seventh intercostal space. The incision is enlarged anteriorly and posteriorly, and strong retractors are inserted. This may provide sufficient room for thorough exploration; but for older subjects it will probably be advisable to divide the superjacent rib (just posterior to the angle) to permit wider retraction. The blood and effused fluid are then removed from the pleura by suction or by gauze.

The injury to the lung may now be seen, and foreign bodies, including pieces of clothing and in-driven bone fragments, removed. Lacerated portions of lung, especially those to which the vascular supply is doubtful, should be excised and all tears in the lung should be sutured.

When gross laceration of a lobe or larger portion of a lung has occurred, a lobectomy may be needed. The hilum is temporarily controlled by a tourniquet and the lacerated lobe beyond is removed. The vessels and bronchi are controlled by the insertion of mattress sutures of catgut and the tourniquet is removed. If the bronchi are closed and no bleeding points discovered, the fringe of lung surrounding the cut bronchi is finally sutured over them.

Drainage should always be used. It has the advantages that it provides an outlet for any further blood and fluid effused into the pleura, it permits the escape of the air in the pleura at the end of operation (which must otherwise be aspirated, or it will cause a considerable degree of surgical emphysema when the patient coughs), it prevents the deleterious effect of a tension pneumothorax should leakage from the bronchi occur subsequent to operation, and drainage is already established if infection should supervene, which it may do even during transport of a patient. Usually it is sufficient to use a tube placed through a stab wound in the ninth intercostal space posteriorly. Drainage in the presence of gross contamination may require resection of a small portion of the ninth rib in the region of the angle. The small incision is firmly sutured around the tube, which is likewise sutured to the skin, a long portion being left outside of the chest. Drainage should not be performed through the wound itself, as it is apt to lead to severe wound infection.

The chest wall is now sutured in layers. Pericostal sutures of strong catgut are first inserted, followed by interrupted sutures in the muscular layers in the chest wall. The intercostal muscles need not be sutured, as the stitches usually tear out. The skin is then sutured.

The chest should be firmly strapped with "Elastoplast" or similar adhesive over a dry dressing, the patient returned to bed and placed in a sitting posture, and the tube connected to a drainage bottle.

Gaping Wounds and Abdomino-Thoracic Wounds.

There are two further injuries the treatment of which should be discussed—namely, gaping wounds of the chest wall and abdomino-thoracic wounds.

A gaping wound in the chest wall too large for suture may be treated by one of two methods. There is the method of packing. The pleural cavity is cleansed of all blood and foreign bodies and then packed with strips of gauze soaked in acriflavine. An air-tight dressing is applied and left for eight days. The lung will then be found adherent to the parieties. If a pleural exudate develops it should be aspirated. A large granulating wound is left, which may need a later skin graft. Alternatively one may treat this wound by pneumopexy, in which the lung is sutured to the margins of the wound thereby shutting off the pleural cavity. If the lung is prolapsed, it should be first reduced by squeezing out air. In late cases, if the lung is prolapsed and necrotic, the necrotic part is excised and the stump is sutured to the wound in the chest wall.

In abdomino-thoracic wounds the prognosis is better if the wound is on the right side than if it is on the left, because on the right side there is the solid liver, as opposed to the hollow viscera on the left side. The treatment will vary according to the type of abdomino-thoracic wound present.

A through-and-through wound on the right side caused by a small object may need only expectant treatment. When the foreign body causing the wound is retained in an inaccessible position, as in the liver, again expectant treatment only is usually required. When a small thoracic wound only is present, but there are signs of grave intra-abdominal mischief, the correct procedure is abdominal exploration by an appropriate abdominal incision. The thorax may require no treatment.

In a case of a severe through-and-through abdomino-thoracic wound with a thoracic wound needing treatment, the thorax should be dealt with first and the upper part of the abdomen approached through the diaphragm. If a retained foreign body needs removal in these cases, it may be approached through the thoracic wound and diaphragm.

The set operation for abdomino-thoracic exploration in these cases is as follows. Firstly, the eighth rib is excised. Then wide retraction is obtained by the cutting of the ninth rib at the posterior angle. The diaphragm is examined, and so are the lung and pleural cavity. The phrenic nerve is crushed as it runs on the pericardium. This causes temporary paralysis, and the diaphragm rises. The wound of the diaphragm is excised and enlarged in the direction of its fibres. Now one is able to examine the left lobe of the liver, the spleen and the stomach. If drainage is required, this may be instituted through the anterior abdominal wall.

Complications of Penetrating Wounds of the Chest.

Infection of a hæmothorax is commonest in penetrating wounds, and usually arises from the wound track or from the missile itself. A closed hæmothorax may be infected from the blood stream or an injured lung. The onset of infection may be sudden, and in some cases it shows itself in a sudden increase of fluid or of gas if the infection is anaerobic. The general condition of the patient becomes worse. There is a rise in the respiratory and pulse rates, but usually little obvious change in the temperature, which may be high in uninfected hæmothorax. In many cases it is impossible to grow organisms from the aspirated fluid, but smears on a slide reveal them. An offensive odour and purplish colour of the fluid denote with certainty an anaerobic infection.

In these cases the dosage of sulphapyridine ("M & B 693") must be increased. All the fluid obtainable from the pleura must be aspirated. The fluid may be found either to become clearer and less turbid or more purulent in successive aspirations. Aspiration should be continued if the fluid becomes clearer; but if it becomes more purulent it will be advisable to perform drainage by rib resection. The rapid formation of gas by anaerobic organisms may cause such dyspnœa as to necessitate intercostal drainage urgently, the patient being too ill for rib resection.

Apart from the occurrence of infected effusions, bronchopneumonia is one of the more common sequelæ of chest wounds.

Massive collapse of the contralateral lung or the upper lobe of the affected lung is apt to occur, just as after operations on the lung in civil life, and may need bronchoscopic exploration to remove obstruction in the affected bronchus.

Pericarditis is not an uncommon complication of infected hæmothorax, even when the pericardium is primarily uninjured. It is a noteworthy fact that gas gangrene usually occurs in the chest wall and not in the lung itself. The lung itself is resistant to infection, and gas gangrene and abscess formation are fairly rare complications.

Late Operations.

Later operations may be required, months or years after the accident, as in military surgery, the commonest being for chronic empyema after an infected hæmothorax. Retained foreign bodies may require removal afterwards, for they may cause pulmonary abscess or repeated hæmoptysis.

COMPLEMENT FIXATION IN INFLUENZA WITH EMBRYO FLUID AS ANTIGEN.[1]

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COMPLEMENT fixation in influenza was first studied by Smith[(1)] in 1936, and a number of papers on the reaction have since been published. A recent review of the work is contained in the paper by Lenette and Horsfall.[(2)] The antigen involved is not the actual virus, but a soluble substance, apparently a protein, which is present in most forms of infective material. Infected mouse lung has been used as a source of antigen, particularly with ferret serum. Lush and Burnet[(3)] found that satisfactory antigens could be prepared from chorio-allantoic membranes infected with virus strains adapted to propagation on this tissue. In view of the high virus titre of the allantoic fluids in chick embryos inoculated by the amniotic route (Burnet[(4)]) we have tested this fluid as an antigen for complement fixation. A similar study by Henle and Chambers[(5)] was reported when our work was nearing completion. Our results confirm their finding that infected allantoic fluid is an excellent antigen, requiring no manipulation beyond a brief centrifugation before use and showing no anticomplementary activity.

It is already known that the complement fixation reaction does not run parallel to virus neutralization. In a study of the serological reactions of a group of volunteers inoculated with influenza A virus, a full account of which experiment was previously published (Burnet and Foley[(6)]) it will be shown that the appearance of complement-fixing antibody is a more sensitive index of recent infection than is a rise in titre of neutralizing antibody.

Technique.

Antigens.

The antigen studied was the pooled extraembryonic fluids (allantoic and amniotic) from at least three eggs inoculated at the twelve-day stage with the standard mouse passage strain "W.S." according to the amniotic technique described by Burnet.[(4)] The fluid was centrifuged for ten minutes

[1] Work carried out with the aid of grants for research on virus diseases from the National Health and Medical Research Council and from Mr. E. Alec Cato.

and stored in the refrigerator. As the egg fluids were sometimes found to increase the apparent hæmolytic titre of complement, the antigen was titrated in detail with complement each day.

Serum.

In the preliminary studies the antigens were tested against pooled influenza-immune adult human serum and pooled influenza-immune monkey serum. Normal monkey serum was included as a control in every test. The human serum was inactivated at 56° C. for twenty minutes and was not anticomplementary in the dilutions used. A higher temperature of inactivation, 60° C. for twenty minutes, eliminated the anticomplementary effects shown by monkey serum when inactivated at 56° C. The human serum from the subjects of the experiments had been inactivated before storage and had been kept in corked tubes for ten or eleven months in the refrigerator.

Complement.

Fresh guinea-pig serum was titrated each day and a dilution was made which contained three minimum hæmolytic doses.

Sensitized Red Cells.

The test dose consisted of 0·1 cubic centimetre of a sensitized 3% suspension of sheep erythrocytes containing five minimum hæmolytic doses of hæmolytic amboceptor per cubic centimetre.

The Test.

Doubling dilutions of serum and antigens were prepared and set up as follows:

Equal volumes (0·1 cubic centimetre) of saline solution, serum dilution, complement dilution and antigen were mixed and incubated for one hour at 37° C., after which one volume of sensitized red cells was added, and the mixtures were shaken thoroughly, placed in the water bath at 37° C. and shaken frequently during half an hour; after this the results were read immediately.

Results are expressed in terms of the initial dilution—that is, a dilution five times as concentrated as the actual dilution in the test. Under these conditions no non-specific effects were ever observed between normal egg fluid and immune human or monkey serum, or between infected egg fluid and normal monkey serum.

In order to determine at what period after inoculation the embryonic fluid contained the maximal amount of complement-fixing antigen, a series of twelve-day embryos were inoculated amniotically with strain "W.S." The specimens of fluid taken after two, three and four days' incubation at 35° C. were tested for virus content by intranasal inoculation in mice and as complement-fixing antigens against pooled immune human and monkey serum. In an abbreviated form Table I gives the essential findings from this experiment. A full series of controls was included in the actual experiments, comprising serum and antigen alone, immune serum with normal egg fluid, and normal monkey serum with each antigen. None of these showed any fixation. Similar tests with immune serum diluted to 1 in 10 gave the same indication that the three-day antigen was slightly more potent than either the two-day or the four-day material.

Changes in the Serum of Volunteers Experimentally Inoculated with Influenza Virus A.

We have previously described the results of a series of experimental inoculations of influenza virus A in human subjects (Burnet and Foley[40]). The following is a brief summary of this work. A group of 15 volunteers received inoculations into the nose and throat of three strains of influenza A virus of progressively increasing virulence, each inoculation being given one week after the preceding one. Blood was taken immediately before the first instillation and seventeen to twenty days after the final one. All samples of serum were titrated for neutralizing antibody content by the chorio-allantoic method against "Melbourne" egg virus. Three subjects whose initial antibody level was low suffered from clinical influenza after the final inoculation. The other subject, D.C., who showed a big neutralizing antibody rise without a clinical attack, also had a low initial level.

The specimens of serum were not studied for complement-fixing antibody until ten or eleven months after they had been obtained; during the interval they were stored in the cold. In the test doubling dilutions of each serum were made and set up against a 1 in 5 dilution of three-day "W.S." allantoic fluid in the presence of one volume of a complement dilution containing three minimum hæmolytic doses. After incubation at 37° C. for one hour the hæmolytic system was added, and the results were read after a further incubation for half an hour. Antigen and serum controls were always included.

In view of the possibility that the egg fluid in the infective material used for the human inoculations might have provided anti-egg antibody, an additional control was provided by the use of fluid from eggs infected with influenza B virus. This virus is antigenically distinct from A, but produces similar lesions in the chick embryo; any difference between the two antigens would then be wholly referable to specific influenza A virus components. In no case was any fixation observed with the B virus fluid. The table contains no data in regard to M.S., and incomplete data of E.W.B. and J.R.C., owing to the exhaustion of one or both samples of serum from these subjects in the course of earlier investigations.

It will be seen from Table II that in all cases in which a rise in neutralizing antibody occurred, a corresponding rise in complement-fixing antibody was shown even in those cases in which the rise in both antibodies was relatively small. The most interesting feature of Table II is that in two instances (F.M.B. and P.R.B.), and probably in a third (J.R.C.), there was no significant rise in neutralizing antibody, but a definite appearance of complement-fixing antibody.

It will be obvious, of course, that with the methods used a considerable absolute increase of neutralizing antibody above an initially high level may not have been recognized, although the same absolute amount produced in a subject with no initial antibody would represent a large rise in titre. We presume that any infection sufficient to provoke complement-fixing antibody would also induce some formation of neutralizing antibody, and the failure to detect this is to be ascribed to inadequacy of technique. Actually a slight rise was recorded in the case of F.M.B., but this was not beyond the limits of experimental error.

TABLE I.

Comparative Activity of Egg Fluid Antigens Collected Two, Three and Four Days after Amniotic Inoculation.

Antigen.	Human Immune Serum 1 : 5; Antigen Dilutions.					Monkey Immune Serum 1 : 5; Antigen Dilutions.					Virus Titre of Antigen.[4]
	2	5	10	20	40	2	5	10	20	40	
Two-day	c[1]	c	—[3]	—	—	c	c	c	c	tr[2]	2×10^6
Three-day	c	c	c	c	—	c	c	c	c	tr	5×10^6
Four-day	c	c	c	±[2]	—	c	c	c	c	tr	5×10^5

[1] c = Complete fixation, no hæmolysis.
[2] ±, tr = Partial hæmolysis.
[3] — = No fixation, complete hæmolysis.
[4] Virus titre expressed as the reciprocal of the concentration giving average Grade II lesions in mice inoculated intranasally with 0·05 cubic centimetre.

Table II.

Complement Fixation in a Group of Volunteers Experimentally Inoculated with Influenza Virus.[1]

Subject.	Neutralizing Antibody Rise.[2]	Complement Fixation.[3]	
		First.	Second.
1. E.L.B.	++	−	±40
2. E.W.B.	++	−	0
3. D.C.	+++	−	±40
4. M.N.	+++	−	±20
5. L.K.	−	−	−
6. G.P.C.	−	−	−
7. F.M.B.	−	−	+5
8. M.C.	−	−	−
9. H.A.M.	±	−	±5
10. M.B.	±	−	±5
11. J.R.C.	−	0	+10
12. W.A.C.	−	−	−
13. P.R.B.	−	−	±20
14. L.B.	−	−	−

[1] The subjects are arranged in order of initial antibody level. Numbers 1 to 4 showed very low antibody, numbers 5 to 12 moderately high antibody level, numbers 13 and 14 very high antibody level.

[2] Neutralizing antibody: "−" indicates no significant rise in neutralizing antibody; "±" indicates a two to five times increase in neutralizing antibody; "+" indicates a five to 50 times increase in neutralizing antibody; "++" indicates a 50 to 500 times increase in neutralizing antibody; "+++" indicates more than 500 times increase in neutralizing antibody. Further details of the antibody responses of these subjects can be obtained from Figures III and IV of the paper by Burnet and Foley.[6]

[3] Complement fixation: "−" indicates complete haemolysis with 1:5 serum; "±" indicates partial fixation at dilution shown, complete fixation at lower dilutions; "+" indicates complete fixation at dilution shown; "0" indicates serum not available for test.

If one examines the results in Table II it will be observed that although there was a wide initial range in titre of neutralizing antibody, four subjects having very low values and two particularly high ones, none of the initial samples which were available for test contained any complement-fixing antibody. In the course of the experiment the majority of the subjects, and all in whom there was any evidence that infection had occurred, showed an appearance of complement-fixing antibody. Since we must assume that the initial virus-neutralizing titre was the residual result of previous infection with influenza virus, our findings provide strong indirect evidence that the complement-fixing titre falls to an undetectably low level much more rapidly than the neutralizing antibody. This is in agreement with the reports of other workers on the subject.

Summary.

1. Allantoic fluid from embryos infected by the amniotic method with influenza virus is a satisfactory antigen for complement fixation tests.

2. In a series of volunteers inoculated nasally with virulent influenza virus A, the appearance of complement-fixing antibody was a more delicate index of infection than a rise in neutralizing antibody.

3. Indirect evidence is given that after infection the titre of complement-fixing antibody falls much more rapidly than that of neutralizing antibody.

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A BIOCHEMICAL INVESTIGATION OF THE TANNIC ACID AND SULPHANILAMIDE TREATMENT OF BURNS.

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The following investigation was prompted by a number of considerations. One of us (A.C.McE.) has been interested for some time in the use of various antiseptics combined with tannic acid in the treatment of burns. For about two years an experimental mixture of tannic acid (4%), gentian violet (1%) and acriflavine (0·1%) was used with considerable satisfaction, and when another antiseptic for experimental use was sought, sulphanilamide was the obvious choice. So far as we were aware, sulphanilamide had not been used extensively in the local treatment of burns.

Colebrook[1] reported 35 cases in which infected granulating areas, produced after burns, were treated by the application of powdered sulphanilamide for one or two days. From only two was a culture of streptococci obtained after two or three days.

Since the completion of this investigation Robson and Wallace[2] have reported the use of a preparation of "Albucid powder", glycerine, cod liver oil and water-soluble sulphanilamide.

A British Ministry of Health instruction to the Emergency Medical Services, dated February 25, 1941, recommends the application of sulphanilamide powder prior to tanning.

Burns are to some extent analogous to infected wounds, and for that reason it is of interest to summarize some of the recently published work in this regard.

Local Application of Sulphanilamide to Wounds.

Nitti[3] applied gauze compresses, soaked in a culture of streptococci, to experimentally made wounds in the dorso-lumbar muscles of rabbits. One gramme of sulphanilamide was applied and the skin was closed. Controls died in twenty-four to forty-eight hours; those treated were still alive in twenty-four to forty-eight hours, and when the wounds had been reopened, the gauze removed and fresh sulphanilamide added, most of the animals survived. Local application of sulphanilamide to the wound never caused irritation; the toxicity was low, and all the sulphanilamide was absorbed and eliminated in forty-eight to sixty-four hours. Legroux,[4] using guinea-pigs, dusted the wounds with sulphanilamide and infected them by inserting a piece of cloth soaked in a culture of *Clostridium welchii* or *Clostridium histolyticum*. Controls died in two or three days; the animals treated with sulphanilamide died after four to seven days, and if the wound was reopened, the cloth removed and new sulphanilamide added, the animal survived.

Jensen, Johnsrud and Nelson[5] treated 41 patients suffering from compound fracture with five to fifteen grammes of sulphanilamide applied locally, and the usual surgical measures were used. The previous infection rate of 27% in their series was reduced to 5%. The concentration in the serum oozing from the wounds was 250 to 600 milligrammes *per centum* (maximum solubility 800 milligrammes *per centum*). Herrell and Brown[6] have obtained significant results by applying to a variety of infected wounds a mixture of three grammes of sulphanilamide and 100 cubic centimetres of saline solution.

Wardle[7] and Eisenhammer[8] have shown that insertion of sulphanilamide into the knee joint cavity causes no reaction, thereby illustrating the drug's blandness.

Hawking[9] took transverse slices of injured muscle, 0·5 centimetre thick, at various distances from a thigh wound

treated with sulphanilamide, and control muscle sections from the opposite thigh; blood was taken from the heart also and the sulphanilamide content was estimated. In the first series of experiments it was found that in six hours solid sulphanilamide was still present in the wound, but after twenty-four hours it had practically all gone. At six hours the concentration in the muscle at a distance of four or five centimetres away from the wound was no greater than elsewhere; the level of free sulphanilamide in the control muscle was also unexpectedly higher than in the blood. In a second series of experiments wounds were made in the standard manner, 0·2 gramme of various compounds was inserted and the wounds were closed. After three days the animals were killed and estimations were made of the drug concentrations in the muscle of the wound, the opposite thigh and the heart blood. Sulphanilamide was rapidly absorbed, so that its influence in the wound was probably negligible after twenty-four hours. Solid sulphapyridine persisted in the wound for seven to ten days (the solubility in 0·85% saline solution at 37° C. is 42 milligrammes per 100 cubic centimetres). Sulphathiazole persisted in the wound over four days, and during this period the concentration in the wounds was higher than that of sulphapyridine, owing to the greater solubility of sulphathiazole (the solubility in 0·85% saline solution at 37° C. is 104 milligrammes per 100 cubic centimetres). The concentration of sulphanilamide in the blood was less (half to one-third) than in the control muscle and greatly diminished ($^1/_{150}$ to $^1/_{200}$) relative to the wound concentration; the concentration of sulphapyridine in the blood was about the same as in the control muscle and diminished ($^1/_{20}$ to $^1/_{40}$) relative to the wound; the concentration of sulphathiazole in the blood was less (about one-half) than in the control muscle and greatly diminished ($^1/_{100}$ to $^1/_{200}$) relative to the wound.

Jensen, Johnsrud and Nelson[49] in two cases, after ten and six grammes of sulphanilamide had been inserted into wounds, found that the highest blood level was reached in about eighteen hours, and the level fell slowly till a trace could be detected after sixty hours. Nitti found a rise in the sulphanilamide level of the blood till about twelve hours and the drug was not completely eliminated until after forty-eight to sixty hours.

Fenner and Bonnin[120] have shown that sulphanilamide applied locally has a pronounced inhibitory effect on the growth in wounds of *Clostridium welchii* and *Clostridium septique*, a slight delaying action on *Clostridium œdematiens* and a probable delaying action on *Staphylococcus aureus*.

Rationale of Local Sulphanilamide Treatment.

The oral administration of sulphanilamide in therapeutic doses results in a level of sulphanilamide in the blood of about 10 milligrammes *per centum*. With the chemical constituents of the blood in equilibrium with the tissue fluid, the local tissue concentration is therefore about 10 milligrammes *per centum*.

Sulphanilamide has a solubility of 800 milligrammes *per centum* and might be expected to reach this concentration in the tissue fluids at the wound surface when applied locally—a concentration many times higher than is obtainable by oral administration. This has been shown to be true by Jensen, Johnsrud and Nelson.[49]

Evidence of Absence of Local Irritation.

The authors have used sulphanilamide locally on a number of occasions in other conditions, without finding any evidence of local irritation, and in burns their experience has been similar.

Sulphanilamide in quantities up to 30 grammes in infected wounds produced no apparent reaction. "Soluseptasine" has been introduced into the knee joint in septic arthritis and into the subarachnoid space in meningitis without any obvious clinical aggravation of the local condition.

In one case in which sulphanilamide was introduced into an extradural abscess, an aseptic meningitis resulted; but this may equally well have been due to the operative disturbance, while the sterility of the cerebro-spinal fluid may perhaps have been due to the sulphanilamide.

Sulphanilamide was applied to a guinea-pig's conjunctiva by insufflation of the powder and by smearing with various bases containing sulphanilamide. The samples of powdered and crystalline sulphanilamide produced no signs of irritation. The viscid base preparations were also non-irritating, except in one instance, when the base was of a high alkaline content.

Preparations Used in this Series.

The preparations used in the cases to be described consisted of a suspension of powdered sulphanilamide (7·5%), tannic acid (4%) in mucilage in one series, and a suspension of sulphanilamide (7·5%) in cod liver oil (25%) and "Vaseline" in a second series. A strength of 7·5% of sulphanilamide in the mixture was chosen on the assumption that if sulphanilamide was absorbed continuously from an open surface over a period of sixty hours, such a concentration would not produce poisoning if the drug were absorbed.

Scope of the Investigation.

Eleven patients were treated with tannic acid sulphanilamide jelly and five with cod liver oil, "Vaseline" and sulphanilamide; the ages of the patients ranged from nine months to ten years. The absorption of sulphanilamide was checked by estimation of the blood level in all the cases.

Method of Treatment.

It was decided to treat the burns by routine cleansing methods before the application of the coagulum, since it was not known whether the sulphanilamide present would of itself be effective. However, after the first few cases had proved that absorption into the circulation did occur, cleansing was to some extent purposely relaxed. A general anæsthetic of ethyl chloride and "open ether" was given in most cases, and the burnt skin was cleansed and rubbed off with tampons soaked in ethereal soap and ether. The jelly kept in a jar was applied to the burn on a wooden spatula and smeared over. The amount of jelly was measured in an ordinary "Lactogen" measuring spoon before use. The progress of coagulum formation was observed at intervals and another thin application was put on later when it was deemed necessary. Drying was hastened by an electric light shock cradle placed over the burnt area. The time taken for the coagulum to form was necessarily recorded approximately. In each case the time for complete separation was recorded. The presence or absence of pain over the injured area and the necessity for after-treatment of an incompletely healed burn were also observed. General treatment for shock was given when necessary.

Method of Blood Sulphanilamide Estimation.

The sulphanilamide level in the blood was estimated by a micro-method. The technique used was as follows:

Capillary blood (0·2 cubic centimetre) was drawn up into a capillary blood pipette and mixed thoroughly in 3·0 cubic centimetres of distilled water. The proteins were precipitated by the addition of 2·0 cubic centimetres of 50% trichloracetic acid. To 2·0 cubic centimetres of the filtrate 0·2 cubic centimetre of 0·1% sodium nitrite solution was added. After the mixture had been allowed to stand for three minutes, two cubic centimetres of alcoholic dimethyl-α-naphthylamine (0·2%) were added. Standard solutions of 0·3 cubic centimetre and 0·6 cubic centimetre of stock sulphanilamide solution were diluted to 100 cubic centimetres. To 0·2 cubic centimetre of each, 1·2 cubic centimetres of water, 0·8 cubic centimetre of trichloracetic acid (50%), 0·4 cubic centimetre of 0·1% sodium nitrite solution and 4·0 cubic centimetres of 0·2% dimethyl-α-naphthylamine solution were added. The colours were compared and calculation was made from the following formulæ:

$\frac{S}{U} \times \frac{3}{1}$ milligrammes *per centum* with the first stock sulphanilamide solution.

$\frac{S}{U} \times \frac{6}{1}$ milligrammes *per centum* with the second stock sulphanilamide solution.

TABLE I.

Treatment with Tannic Acid and Sulphanilamide Jelly.

Case No.	Nature of Accident.	Site and Degree of Burn.	Previous Treatment and State of Burn.	Area of Burn in Square Inches.	Duration of Burn in Hours Before Treatment.	Amount of Paste. (Ounces.)	Time Taken for Formation of Coagulum in Hours.	Time for Complete Separation in Days.	Blood Sulphanilamide Levels in Milligrammes *per Centum*.	Time for Complete Healing; Complications, Pain *et cetera*.
I	Scald from boiling water.	Arm, 1st and 2nd. Chest, 3rd Face, 1st and 2nd.	"Tannafax"; soiled, infected.	33·0	3	1·0	24	20	Hours: 7 12 21 28 32 5 —[1] 3 1·5 —	24 days; coagulum cracked in axilla after four days and slight infection of small area occurred.
II	Scald from boiling broth.	Hands, 1st, 2nd and 3rd. Forearms, 1st, 2nd and 3rd.	Clean.	22·0	21	0·75	24	7	Hours: 6 11 16 20 5 3 trace —	Fingers covered; no gangrene, no contractures; 8 days.
III	Scald from boiling water.	Forearm, 1st, 2nd and 3rd. Thigh, 1st, 2nd and 3rd.	Soiled.	22·0	48	1·0	24	8	Hours: 7 18 4·74 —	9 days.
IV	Burn from ignited kerosene.	Back, 1st, 2nd and 3rd.	Clean.	50·0	3	1·25	18	12	Hours: 5 10 15 21 30 35 4·5 4·1 3·2 2·4 trace —	15 days.
V	Sunburn.	Back, 1st and 2nd. Shoulders, 1st, 2nd and 3rd.	Olive oil; infected.	48·0	168	1·0 and 1·0 2 applications	18	10	Hours: 6 10 18 24 30 4·5 2·4 2·3 trace —	12 days.
VI	Sunburn.	Chest, 1st and 2nd. Back, 1st and 2nd.	Infected.	57·0	96	0·75 and 0·5 2 applications.	36	10	Hours: 6 10 16 20 24 28 30 4·1 3·7 3·4 3·0 1·9 trace —	11 days. Coagulum long in forming.
VII	Methylated spirits burn.	Chest, 1st and 2nd. Back, 1st and 2nd. Shoulders, 1st and 2nd.	Olive oil, sodium bicarbonate and tannic acid jelly, soiled, clean.	40·0	7	1·0 and 1·0 2 applications.	16	24	Hours: 5 10 14 18 24 28 34 3 4 2·5 trace trace trace —	38 days. Crack in axilla allowed infection to occur; complete healing with zinc oxide ointment.
VIII	Scald from boiling water.	Buttock, 1st and 2nd. Back, 1st and 2nd.	Olive oil; soiled and infected.	53·0	6	0·5 and 0·5 2 applications.	12 to 18	18	Hours: 5 10 20 25 30 2·5 3·5 1·5 trace —	24 days.
IX	Sunburn.	Back, 1st and 2nd.	"Vaseline"; infected, clean.	32·0	24	0·5	12 to 18	9	Hours: 5 10 15 20 3·2 2·1 trace —	11 days.
X	Scalded by burning fat.	Foot, 1st and 2nd.	Clean.	5·4	2	0·5 and 0·5 2 applications.	12 to 18	20	Hours: 10 18 2·25 —	20 days.
XI	Sunburn.	Back, 1st and 2nd.	Soiled, infected.	56·0	124	1·0	12	5	Hours: 6 10 15 2 1·5 —	6 days.

[1] Error in collecting blood sample.

Blood samples of 0·2 cubic centimetre were taken from a prick in the most convenient digits. The first was taken approximately four to six hours after the application of the paste, and further samples were taken at approximately similar intervals until no sulphanilamide was present in the blood as determined by colorimetric estimation. The samples, except in one or two instances, were taken by one of the authors to avoid error, and the estimations were all made by a micro-method by the one experimenter (J.deV.). The personal error was thereby reduced to a minimum.

Results.

Although the investigation was designed primarily as a quantitative estimation of the action of sulphanilamide when applied to a burnt surface of the body, the clinical progress and recovery of the patient were noted in relation to the treatment. The data and figures for the burns treated with tannic acid and sulphanilamide paste are listed in Table I.

According to the findings of Hawking,[9] the concentration of sulphanilamide on the surface of a wounded area is much greater than the concentration of the drug absorbed from it into the blood stream. In every case in our tannic acid series absorption of sulphanilamide into the general circulation occurred; thus the assumption was that a fairly high concentration of sulphanilamide was present in the tissue fluids and exudate in the burnt surface area in amounts that could not possibly be attained by oral administration. The absorption into the circulation depended on the concentration of the sulphanilamide in and on the absorbing surface, so that the blood levels of sulphanilamide reflected the concentrations of the surface sulphanilamide. Our results showed a rise of the sulphanilamide concentration, followed by a fall to zero in thirty-six hours. The graphs of sulphanilamide concentration against time in hours showed that sulphanilamide absorption was irregular, both in quantitative and in time values. Obviously from our results these fluctuations were related to the area of the burn and the quantity of tannic acid and sulphanilamide paste applied and the time taken for the coagulum to form. As soon as the coagulum of tannic acid had formed, there was no absorption of sulphanilamide. The conclusion therefore was that the tannic acid "bound" the sulphanilamide as soon as a firm coagulum had formed; but while the coagulum was forming, the drug was acting with increasing effect up to a point when the paste began to enter upon the process of firm coagulation. On the attainment of complete coagulation no further action of the drug occurred (Figure I).

The highest concentration of sulphanilamide in the blood was 5·0 milligrammes *per centum*. As we were using a concentration of 7·5% sulphanilamide in our preparation, it was obvious that a 10% or even a 15% concentration of the drug could probably have been used with safety. Moreover, by a combination of more diffusible sulphanilamide compounds (sulphanilamide and sulphathiazole) with less diffusible forms (sulphapyridine and "Proseptasine"), it may be possible to produce a combination which will produce a high initial concentration of the former and a prolonged action of the latter. The data and figures for the burns treated with cod liver oil and sulphanilamide mixed with "Vaseline" are given.

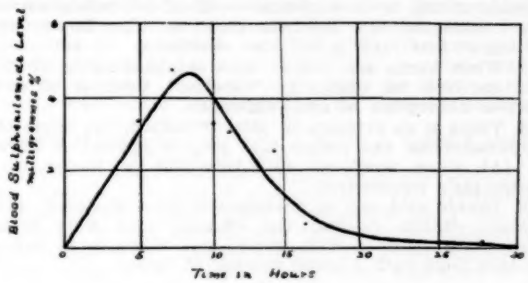

FIGURE I.

Composite graph, showing average blood sulphanilamide levels at various fixed periods in hours after application of sulphanilamide coagulum.

As has already been mentioned, Robson and Wallace,[10] using a preparation of "Albucid powder", glycerine, cod liver oil and water-soluble sulphanilamide, have obtained good results in a small series of cases treated for burns. In our series, however, the absorption of sulphanilamide was negligible and led to the assumption that an oily suspension almost completely "bound" the sulphanilamide and prevented its absorption from the surface tissues into the circulation. In all cases there was subsequent infection of the burn and prolonged and slow healing. Furthermore, reapplication of dressings was most troublesome and painful.

Summary.

1. A biochemical investigation has been made of the absorption of sulphanilamide from the surface of burns, (*a*) when combined with a tannic acid coagulum jelly,

TABLE II.

Treatment with Cod-liver Oil, "Vaseline" and Sulphanilamide.

Case No.	Nature of Accident.	Site and Degree of Burns.	Previous Treatment and State of Burn.	Area of Burn (Square Inches.)	Duration of Burn Before Treatment. (Hours.)	Amount of Paste. (Ounces.)	Blood Sulphanilamide Levels in Milligrammes *per Centum*.	Time for Complete Healing; Complications, Pain *et cetera*.
I	Scald, boiling tea.	Arm, 2nd. Shoulder, 2nd.	Acriflavine; infected, soiled.	16	18·0	1·0	First 24 hours: slight trace; second 24 hours: absent.	30 days; granulations developed.
II	Scald, boiling water.	Arm, 1st and 2nd. Thigh, 1st and 2nd. Chest, 1st and 2nd. Abdomen, 1st and 2nd. Vulva, 1st and 2nd. Buttock, 1st and 2nd.	Nil; clean.	One-third of body surface (child aged 2½ years).	1·0	2·0	First 24 hours: slight trace; second 24 hours: absent.	30 days; granulations; epithelialization incomplete.
III	Ignited celluloid burn.	Forearm, 2nd and 3rd. Hand, 2nd and 3rd.	Olive oil; soiled, infected.	16	5·5	0·5	Not detected.	40 to 60 days; much œdema above burn; contracture of fingers (25th day).
IV	Scald, boiling water.	Forearm, 2nd. Hand, 2nd.	Nil; clean.	12	4·0	0·5	Not detected.	20 days; granulations.

and (*b*) when combined with an oily base such as cod liver oil and "Vaseline".

2. Further evidence of the blandness of sulphanilamide when applied in solid form to a delicate surface is shown in the local application of the drug to the conjunctival sacs of guinea-pigs.

Conclusions.

From the investigation of this small series of cases it is possible to draw the following conclusions:

1. Sulphanilamide (7·5%) in tannic acid (4%) jelly has similar coagulum-forming properties to tannic acid jelly.

2. Sulphanilamide absorption continues for thirty hours, quickly rising to a maximum level of 5·0 milligrammes *per centum* in the blood in eight or nine hours, and falling at first rapidly and then slowly.

3. When burns are treated with sulphanilamide (7·5%) and cod liver oil (25%) in "Vaseline", there is no comparable absorption of sulphanilamide.

4. There is no evidence of local irritation, (*a*) when the sulphanilamide and tannic acid jelly is applied to burns, or (*b*) when powdered sulphanilamide is placed in a guinea-pig's conjunctiva.

5. Tannic acid and sulphanilamide jelly produced satisfactory clinical results; but whether they were better than those obtained with tannic acid alone could not be decided from such a small number of cases.

Acknowledgements.

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A NOTE ON THE PREPARATION AND USE OF M AND N TESTING FLUIDS.

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The preparation of M and N testing fluids was undertaken in order to determine the distribution of M and N factors in the blood of a number of white Australians and of Australian aborigines. The latter will be dealt with in another report.

For an account of M and N agglutinogens Wiener (1939) may be consulted; Boyd (1939) also summarizes the available data and gives a full bibliography.

Methods.

Specimens of anti-M and anti-N absorbed serum were kindly sent to us by Dr. K. Landsteiner and Dr. W. C. Boyd, of the United States of America. Through the courtesy of Dr. E. T. Brennan, Director of Public Health, New Guinea, Major C. Backhouse procured for us some specimens of unabsorbed serum, which had been obtained from Dr. G. L. Taylor, of London. By means of these sera members of the laboratory staff were typed to serve as permanent donors of blood of types M, N and MN. These specimens of serum were later available also as standards with which to compare the quality of our own absorbed serum.

Immunization of Rabbits.

Red blood cells for immunizing rabbits were taken weekly from OM and ON donors and stored at 4° C. in the glucose-citrate solution of Rous and Turner (1916). Before injection they were washed twice and resuspended in a suitable volume of physiological saline solution; the volume injected was measured as packed red cells. Three groups, each of four to eight rabbits, were injected as follows. Half the rabbits in each group were given OM or ON red cells.

1. Small Doses. Injections of 0·25 to 0·5 millilitre of packed red cells were given intravenously for four or five consecutive days, followed by ten days' rest. The first injection after a rest was given subcutaneously. It was found necessary to give 28 injections before antiserum of high titre was obtained.

2. Large Doses. Injections of 15 millilitres of red cells were given intravenously, followed at intervals of one week by 15 millilitres in divided doses, 7·5 millilitres being given subcutaneously in the morning and 7·5 millilitres intraperitoneally in the afternoon of the same day. Three or four injections were given, amounting to 45 to 60 millilitres of packed red cells.

3. Injections of Cell Stroma. Cell stroma was prepared by mixing 100 millilitres of red cells with 900 millilitres of a 0·2% solution of sodium chloride in distilled water. After the mixture had stood overnight at 4° C., the centrifugated deposit was washed in physiological saline solution and resuspended in an equal volume of physiological saline solution. Four weekly injections of 10 millilitres of this suspension of disintegrated red cells, amounting to that derived from 150 millilitres of packed red cells, were given subcutaneously and intraperitoneally.

Absorption of Agglutinins.

The serum was first heated at 56° C. for thirty minutes. A_1M or A_1N red cells were generally used for absorption. The proportion of serum to red cells was generally suggested by a preliminary trial, in which the serum was diluted 1 in 5, 1 in 10 and 1 in 20 and absorbed for thirty minutes at room temperature with one-quarter, one-third and one-half volume of twice-washed packed red cells. It was not always possible, however, to obtain satisfactory testing fluids by means of only one absorption.

The absorbed serum, undiluted and diluted from 1 in 2 to 1 in 32 or higher, was then titrated in test tubes (8·0 by 75 millimetres) with equal volumes of 1% suspensions of M, N and MN cells. The results were read after one hour at room temperature. Further absorption was not carried out if the difference between the highest dilution at which heterologous agglutination was seen microscopically and the highest dilution at which homologous agglutination was seen with the naked eye was wide enough to allow heterologous agglutination to be eliminated by dilution.

For example, anti-N serum was diluted 1 in 10 and absorbed with A_1M red cells. After absorption it caused strong agglutination of N cells, but slight agglutination of M cells also. This latter (heterologous M) agglutination was still visible microscopically when the absorbed serum was further diluted 1 in 2, but not when it was diluted 1 in 4. Weak homologous N agglutination was visible to the naked eye, but agglutination of MN cells was hardly visible, at a dilution of 1 in 32. Strong agglutination of both N and MN cells was seen at a dilution of 1 in 16. The serum was used for testing purposes at a dilution of 1 in 4.

We therefore agree with Olbrich (1936) and with Taylor and Ikin (1939) that complete elimination of heterologous agglutinins by absorption may be unnecessary.

Technique of Typing.

After satisfactory testing fluids had been prepared, three methods of typing were compared by typing 100 specimens of blood by each method, namely: (i) in tubes, (ii) on "Army" tiles (Bryce and Jakobowicz, 1941) and (iii) on glass slides (three inches by two inches) each divided by paint into eight squares. In order to avoid any confusion separate slides were used for M and N testing fluids. Equal volumes of testing fluid and a 1% suspension of red cells were measured by dropping them from standardized Pasteur pipettes (20 drops per millilitre); a separate pipette was used for each testing fluid, and one pipette, which was washed with physiological saline solution between specimens, was used for all the red cell suspensions. The tubes were allowed to stand at room temperature for one hour, the tiles and slides for thirty minutes. Agglutination was not observed on any slide, kept in a moist chamber for thirty minutes, on which it had been absent at fifteen minutes. Slides on which agglutination could not be seen by the naked eye were examined microscopically.

The results obtained by each of the three methods were the same; but for reasons discussed below, subsequent typing was done on glass slides. On these specific agglutination was usually apparent after five minutes, at which time a preliminary reading was taken; a final reading was taken with the naked eye after fifteen minutes.

Results.

The best antiserum was obtained from rabbits of the first group, which received small doses of red cells. Titres were 3,200 to 6,400 in rabbits injected with M cells, 6,400 to 12,800 in those injected with N cells. Several satisfactory specimens of antiserum were prepared, having after absorption a titre of 8 to 16, not including the dilution at which the serum was absorbed. Less satisfactory serum was prepared from rabbits which received large doses of red cells or which received cell stroma. It was hoped that antiserum prepared with cell stroma might be more specific than that prepared with whole cells, and that cell stroma might effect more satisfactory absorption than whole cells. However, no advantage in these respects was derived from the use of cell stroma, although it was agglutinated specifically by the corresponding anti-M or anti-N absorbed serum, and it also effected specific absorption of the corresponding agglutinins.

Of the 1,000 specimens of blood examined, 434 came from members of the Red Cross Society's transfusion service; these persons were selected as having the most suitable blood for the preparation of pooled serum for therapeutic use (Jakobowicz and Bryce, 1941). The proportions of the groups in this series were therefore rather different from those found in unselected white Australians. The remaining 566 were Army recruits and were unselected as regards blood grouping.

The results are shown in Table I, which is presented in the form given by Taylor and Ikin (1939). The agreement between the observed numbers and those expected for a homogeneous population according to the theory of Landsteiner and Levine (1928) that heredity of M and N depends upon a single pair of allelomorphic genes, was tested by means of the χ^2 test, with the formula $\chi^2 = \frac{(b^2-4ac)^2(a+b+c)}{(2a+b)^2(b+2c)^2}$, where a, b and c are the observed numbers (not the frequencies or percentages) in the types M, MN and N respectively. The probabilities corresponding to the values so obtained for χ^2 for one degree of freedom were derived from Fisher's table of the distribution of χ^2. The results for each group and for the whole series were treated in this way, and gave in all cases values of P large enough to indicate that the differences between the observed and expected figures could be due to sampling errors alone.

Of the 1,000 specimens of blood examined, 306 were of type M, 217 were of type N and 477 were of type MN, giving the following gene frequencies: $m = \sqrt{M} = 0{\cdot}5532$ and $n = \sqrt{N} = 0{\cdot}4658$, and $m + n = 1{\cdot}019$. According to the theory of Landsteiner and Levine, $m + n$ in a homogeneous population should be equal to one. In our sample the difference, $D = 1 - (m + n) = 0{\cdot}019$ and the standard deviation of D is 0·0158. The difference D is therefore only 1·2 times its standard deviation, and no departure from the expected frequencies is shown.

That the testing fluids were free from contaminating a agglutinin is shown by the distribution of M, N and MN factors within the A B O groups, which indicates that there was no simultaneous significant excess in the proportion of the heterozygote MN within the groups A and AB.

TABLE I.

The Distribution of Types M, N and MN within the Blood Groups O, A, B and AB.

Series.	A, B, O Groups. (Number and Percentages.)	Numbers and Percentages in Types of MN System.			χ^2.	Probability. (P.)
		M.	MN.	N.		
Selected specimens. (Red Cross Society donors.)	O 124 (28·6)	34 (27·4)	66 (53·2)	24 (19·4)	0·63	0·43
	A 193 (44·5)	59 (30·6)	90 (46·6)	44 (22·8)	0·73	0·40
	B 91 (21·0)	21 (23·1)	40 (44·0)	30 (33·0)	1·15	0·28
	AB 26 (6·0)	7 (26·9)	17 (65·4)	2 (7·7)	3·33	0·07
Total	434	121 (27·9)	213 (49·1)	100 (23·0)	0·11	0·74
Unselected specimens. (Army recruits.)	O 308 (54·4)	101 (32·8)	146 (47·4)	61 (19·8)	0·39	0·53
	A 209 (36·9)	67 (32·1)	97 (46·4)	45 (21·5)	0·79	0·38
	B 30 (5·3)	9 (30·0)	13 (43·3)	8 (26·7)	0·53	0·47
	AB 19 (3·4)	8 (42·1)	8 (42·1)	3 (15·8)	0·17	0·68
Total	566	185 (32·7)	264 (46·6)	117 (20·7)	1·62	0·20
Total specimens (1,000).	O 432 (43·2)	135 (31·2)	212 (49·1)	85 (19·7)	0·012	0·92
	A 402 (40·2)	126 (31·4)	187 (46·5)	89 (22·1)	1·53	0·22
	B 121 (12·1)	30 (24·8)	53 (43·8)	38 (31·4)	1·75	0·19
	AB 45 (4·5)	15 (33·3)	25 (55·5)	5 (11·1)	1·28	0·26
Total	1,000	306 (30·6)	477 (47·7)	217 (21·7)	1·47	0·22

Discussion.

Although Wiener (1939) states that any technique used for ordinary blood grouping is suitable for M-N tests, he, in common with several other authorities cited by Thomas (1937), deprecates slide agglutination. The drying which takes place on the surface of the slide may prevent the results from being read after twenty or thirty minutes, and in this way late agglutination may be missed unless the slide is placed in a moist chamber. Our tests were originally made in a moist chamber, but after we were so fortunate as to prepare testing fluids which caused strong specific agglutination in five or ten minutes, we abandoned its use. We adopted slide agglutination because of the personal preference of one of us (R.T.S.) who prepared the testing fluids.

It seems important to draw attention here to the stringent safeguards which would be necessary if the determination of M and N blood types were to be put to forensic use in Australia. They are necessary because no verification of the result of an individual test is possible, owing to the fact that iso-agglutinins for M and N rarely occur naturally in the serum as they do for A and B.

The following minimal technical requirements are endorsed by the Committee on Medico-legal Blood Grouping Tests of the American Medical Association (Hektoen *et alii*, 1937) and are touched on by Thomas (1937) in his discussion of the model law which he has proposed for adoption in Great Britain. The technical difficulties and the necessary controls are discussed by Taylor and Ikin (1939).

The test should be undertaken only in a laboratory where permanent donors of M, N and MN types are available as controls. One control or more of each type should be included in each test. Separate specimens of anti-M and anti-N serum from three rabbits should be used, and the person undertaking the test should become familiar by frequent use, with each testing fluid (which, preferably, he should have prepared himself). The blood, if received into physiological saline or citrate solution, should be examined on the day on which it is taken. If it cannot be examined immediately, it should be taken aseptically and kept chilled in the sterile glucose-citrate solution of Rous and Turner, containing 0·001% (1 in 100,000) of "Merthiolate". We have had no experience with clotted blood, which is recommended by Wiener. Specimens taken aseptically in glucose-citrate solution and packed in a thermos flask containing ice may be transported satisfactorily for several days during summer. Red cells have been preserved satisfactorily in glucose-citrate solution at 4° C. for as long as three weeks. If the reactions are not definite the tests should be repeated on fresh specimens of blood; if they remain doubtful it is essential to state that no definite conclusion can be reached.

Summary.

1. In our hands, the most satisfactory antiserum against human M or N factors was prepared in rabbits by small daily injections of OM or ON red cells.

2. Satisfactory testing fluids were prepared from some only of the available specimens of antiserum; the best technique for the absorption of the heterologous agglutinins was decided upon as a result of separate experiments with each individual specimen of antiserum.

3. The blood of one thousand white Australians has been typed, with results in conformity with the theory of Landsteiner and Levine.

4. The essential safeguards in the application of M and N blood typing to medico-legal work are discussed.

Acknowledgement.

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IMPROVED METHODS OF MENTAL NURSING.[1]

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Whilst it has long been recognized that improved methods in mental nursing are possible, the exact form they should take has been uncertain. The administration of mental hospitals in all countries is more or less standardized, and the precautions to be taken in the various forms of mental disease are much the same in all text-books of psychiatry, no matter what the nationality of the authors.

The late Dr. Norton Manning, Inspector-General of Mental Hospitals in New South Wales from 1876 to 1898, stressed time and time again in his numerous writings the necessity for mental hospitals to be made as home-like as possible. During the war of 1914-1918 it was found that many of the strict precautions previously taken for the safe keeping of patients in mental hospitals in England could be relaxed. The establishment of psychiatric clinics, such as Broughton Hall in Sydney, showed that many patients with acute mental diseases could be nursed quite satisfactorily in open wards. Furthermore, the establishment of psychiatric out-patient departments in general hospitals showed that many patients who in mental hospitals would require closed ward treatment and much observation, could be quite safely treated as out-patients.

During recent years it has become recognized that insane patients are in reality patients suffering from one of the many forms of mental disease which more often than not can be successfully treated. Hence the present conception of mental hospitals is that they are hospitals for the treatment of patients, and not asylums for the treatment of dangerous lunatics. But just as in medicine and surgery risks must be run in treatment (witness the mortality rate after appendicectomy), so too are risks unavoidable in the treatment of mental patients. For example, the need of continual observation to prevent patients suffering from acute melancholia from committing suicide is stressed by all authorities. But the experience of years has been that no matter what precautions are taken, suicides are inevitable in mental hospitals. The recognition of this fact has led to a modern aphorism in mental hospital administration: "Without many escapes and a few suicides no cures are possible."

In 1937 I decided to see whether it was possible to combine the experiences gained at Broughton Hall and in the psychiatric out-patient department at the Newcastle

[1] Read at a meeting of the Section of Psychiatry, Neurology and Neuro-Surgery of the New South Wales Branch of the British Medical Association on June 19, 1941.

General Hospital (New South Wales) in the administration of this hospital. Kenmore is 130 miles from Sydney and four miles from Goulburn. It was opened in 1895 on an estate of 1,100 acres. For years past the hospital has been justly famous for its beautiful grounds and also for its extensive orchards and farms.

The average patient population is about 650 males and 520 females. There are nine male and eight female wards, which, with one exception, are modern brick buildings. The admission wards are well separated from the main hospital buildings. Until recent years most of the patients admitted were transferred from other hospitals, but today the number of those directly admitted totals over 150 a year. In 1937, 170 males and 40 females were in open wards.

For years past I have been trying to see hospitals from the patients' point of view. Intelligent patients all point out that in both general and mental hospitals patients lose their individuality and become nothing else but cogs in a vast machine. In general hospitals especially tidiness and routine are of more importance than the comfort of patients. They say that a tidy bed is of more importance than the comfort of the patient in it, whilst in many hospitals patients are awakened in the early hours of the morning to be washed, and cannot go to sleep again. Female patients criticize the bare, utilitarian furnishing of wards, and bemoan the absence of mirrors and all home-like touches. I always thought that the risk of sepsis was an insuperable barrier to curtains, mats and such-like in a ward, until I saw the Sacred Heart Hospital at Young, New South Wales, where even the labour ward is furnished more in the style of a comfortable room in a private home than as a hospital ward. If through the genius of Mother Justinian, the foundress of the hospital, such wards were possible at Young, they should be equally successful in other hospitals and especially in mental hospitals.

In 1937, with the full approval of Dr. J. A. L. Wallace, Inspector-General of Mental Hospitals in New South Wales, I decided to furnish the female admission ward in this hospital in the style of a private home, eliminating as far as possible the traditional mental hospital furnishing. The ward was first thoroughly renovated, and by a rearrangement of rooms the accommodation for patients was increased from 36 to 46. The whole ward was painted in bright pastel shades, a different colour for each room. A benefactor, who insisted on remaining anonymous, donated almost sufficient modern furniture to furnish the ward. The usual bare appearance of all hospital beds was overcome by the use of gold or aluminium paint, whilst many of the beds were provided with special dimity quilts, pillow shams, mosquito curtains and even eiderdown quilts. Mirrors, mats, numerous flower vases, and in one dormitory a combination chest of drawers for each patient, comprised the main articles of furniture used. Through the generosity of my predecessor, Dr. C. G. Moffitt, the ward had been liberally supplied with framed pictures. The nursing staff provided window curtains for most of the large rooms. "Rubberoid" was used as a floor covering in the corridors.

Revolutionary changes were made in the single rooms. The traditional function of single rooms, like that of the "strong rooms" in general hospitals, is for the seclusion of violent, refractory and troublesome patients. In these rooms the windows are protected by wooden shutters, only narrow strips of glass are in the door, whilst no furniture, in many cases not even a bedstead, is allowed. If the walls are padded we have the famous "padded cell", which has long since gone out of date. Once single rooms were dark and forbidding; but now, as the result of better planning, they are much brighter than they used to be.

The conception that these rooms should be for privileged and not for refractory patients occurred to Matron Miller at this hospital some years ago. At her instigation the relatives of a few female patients provided wardrobes, chests of drawers, carpets, curtains and such-like for them in single rooms. The success of such rooms was so spectacular that I decided to furnish five of the seven single rooms in the ward, keeping the other two for refractory patients. Two were furnished by the nursing staff, the other three by relatives of patients. The shutters were removed and curtains were provided in their stead. The completed rooms were furnished even more elaborately than bedrooms in most private homes. The patients in them were allowed to display any ornaments, photos and such-like that they cared to. No precautions were taken against suicide, save of course the most obvious ones. The rooms for refractory patients were made as bright as possible, and bedsteads and lockers were provided. With increasing experience, more and more furniture was cautiously added, until now they are but little inferior to the others. In fact, the occasions on which the rooms must be stripped to deal with refractory patients are becoming very rare.

All patients in the ward were encouraged to wear their own private clothing. If clothing had to be provided for patients, steps were taken to ensure that it corresponded to that worn by women in the outside world. Even foundation garments and knitted woollen bed jackets were secured. The effect of such clothing on patients is reflected in the fact that the patients in the ward are rarely destructive to their clothing.

Every effort was made to make the ward as self-contained as possible. All laundry facilities, including an electric iron, were provided, and the patients were encouraged to do their own private washing—a concession that has been greatly appreciated. All cooking is done in the ward and even supper is provided. Thus the traditional mental hospital atmosphere has been removed and, except for the locked doors, the ward is akin to a good boarding house.

During the many months it took to develop the ward, three of the senior nurses were sent to Broughton Hall to gain further experience, and a female occupational block was established. New methods were tried very cautiously. The first thing done was to relax the precautions against suicide.

It happened that there was in the ward a patient, aged thirty-five years, who had been regarded for months as actively suicidal. She promised me not to commit suicide. I took her at her word, relaxed all precautions and allowed her to go for walks in the grounds with another female patient. She went home perfectly well within a few weeks and has remained well since.

We have learned that if a patient suffering from melancholia promises not to commit suicide, his word can be taken. So far we have met with no exceptions to this rule.

A man, aged forty-eight years, was regarded as actively suicidal in another hospital. He was admitted here as a voluntary patient over twelve months ago, and a day or two after his admission he promised me not to commit suicide. I then allowed him to work in the grounds without the slightest observation. He recovered rapidly, went home and has remained well since.

Indeed our confidence has so grown with increasing experience that we now use little or no observation in the treatment of patients with acute melancholia, even including those with acute agitated melancholia.

We have also discovered that a few melancholics, especially those of the mild involutional type, when granted independence of action, often forget their own worries in caring for other patients. I have seen more than one disinterested patient regain interest in things when she has been asked to take another patient for a walk in the grounds. Dr. Kinna, the medical superintendent of the mental hospital at Stockton, New South Wales, informs me that he has noticed the same thing among his patients at Stockton.

With the relaxation of strict observation the atmosphere of the ward changed. The nurses, no longer afraid that harm would befall their patients, lost their fears and became more natural. The patients, even the most suspicious of them, no longer under close observation day and night, lost their suspicion of the staff and hospital and were only too willing to cooperate in the work of the ward. The result was that not only had the nurses more time to study their patients, but the work of the ward could be done efficiently with five nurses for 46 patients instead of six nurses for 36 patients as formerly. The reduction of nursing difficulties in such a ward is well shown by the fact that patients difficult to feed are

becoming increasingly rare, whilst no patient has been tube fed in this hospital for over three years.

Our best results have been obtained in the treatment of patients with acute toxic insanity. The patients are acutely ill and may have heart disease, pneumonia or any other severe bodily illness. *Delirium tremens* is the best known of an extensive group. Years ago the disease was termed acute delirious mania, and was considered to have a grave prognosis. The patients are noisy, restless, confused, and, in spite of large doses of sedatives, sleepless. They will not stay in bed, and in general hospitals are restrained by a restriction sheet. They are usually rational by day and noisy at night. The severity of the disease varies enormously. It may be only transient, as it so often is with feverish children; it may be so severe that the patient dies of exhaustion after a few hours. The prognosis is usually that of the disease causing the condition.

Years ago I noticed that the less the patients were interfered with, the quieter they were. This fact is well known in the treatment of cerebral irritation. It is also clear that in many patients, especially those with *delirium tremens*, the acute restlessness is due to the terror caused by vivid and terrifying hallucinations of sight and hearing. If such patients are put in a restriction sheet, as they so often are, they cannot escape from their hallucinations and are likely to die of sheer terror. They are terrified of the dark, and if they are in dark single rooms they make desperate efforts to escape, even to the extent of trying to break down the walls. "Padded cells" were undoubtedly introduced to prevent such patients from injuring themselves. The patients are difficult with food, imagining that it is poisoned. This is certainly due to an illusion of taste caused by the dryness of the mouth and the heavily coated tongue.

The treatment adopted has been based on our conception of the disease. Except for actual nursing requirements, the patients are left severely alone. During the day they may be kept in a single room, or in bed in a dormitory, or even on a bed in the airing court, according entirely to where they are the least restless. If they will not stay in bed, or if they continue to bang at the door of their single room or to wander restlessly around it, it has been found that to let them wander round the ward is far less exhausting for them. In such cases they usually curl up on a chair in some corner of the ward and doze off to sleep. The tongue is cleaned frequently with glycerine and borax, and they are fed liberally with fluids, milk, egg flips and as much water as they can drink. Many of these patients if left alone will feed themselves. The only sedative given is paraldehyde in two drachm doses at night. This can be repeated in four hours if necessary. The patients sleep in a lighted room or in a dormitory, again according to where they are the least restless.

The results of this treatment, especially in the type of ward I have described, are a source of the greatest satisfaction to us. Deaths in this disease must be inevitable; but the fact that the only two patients suffering from cardiac delirium whom I have ever seen to recover have recovered under this régime has convinced me of its value. The reason why I have abandoned all sedatives except paraldehyde in this disease is that it is my opinion that not only are many of these cases precipitated by sedatives, especially morphine, but that the symptoms are accentuated by them.

The willing cooperation of patients in the ward has made all other forms of treatment easier. Thus there is now little difficulty in inducing patients to go to the occupational therapy room, whilst their terror of the "Cardiazol" shock treatment is becoming less pronounced. The effect, too, of the ward on the nursing staff has been equally great. The nurses have become much keener and take far more interest in the patients. This is well shown by their generosity in donating furniture to the ward, and also by the fact that they frequently take patients out for the day. The relatives of the patients in the ward are most grateful and seldom complain, whilst the townspeople seldom raise any objection to their relatives being admitted here, even as voluntary patients, in the early stages of their illness.

Whilst this type of ward is a distinct advance in mental hospitals, its development caused much worry and anxiety. We had few precedents to go on and had to rely on our own experience as to what liberties to give patients. Many errors of judgment were made and are still being made. Except for young mentally defective girls, who can never be trusted, dements, who are unable to appreciate such refinements, and advanced deteriorated epileptics, all types of patients were tried in the ward. It was soon found that patients suffering from acute mania and restless senile melancholics were not suitable for treatment under such conditions.

The most troublesome patients in this as well as in other wards are inebriates. A great majority of patients certified under the *Inebriates Act* are model patients; a minority give more trouble than the rest of the patients put together. They do not tell the truth, their word cannot be trusted, they abuse privileges and create discord among the other patients. It is interesting to note that such patients cannot appreciate privileges that normal and insane persons can. The argument that mental hospitals are not suitable for the treatment of inebriates is probably right, but only for the reason given above.

Moral imbeciles were seldom a success in the ward. Such patients give trouble wherever they are, and refinement is wasted on them. They behave themselves for only as long as it suits them, and the only way they can be managed satisfactorily in a mental hospital is by strict discipline in a refractory ward. Most habitual criminals are recruited from the ranks of such patients, and those who think they can reform them by kindness, sympathy and understanding must have had very little experience with them. Thus the two classes of patients, inebriates and moral imbeciles, who protest the most about their detention in mental hospitals, failed where mental patients succeeded.

We also found that some patients were too comfortable in the ward and tended to fall into a groove, making no effort to help themselves. A change to another ward usually improved their condition.

The lessons taught us by this ward have been gradually extended to all the other wards and especially to the male admission ward, where the results have been equally good. Male patients can be given more liberty than female patients, and even those recently admitted with a history of attempted suicide have been allowed to work alone in the grounds. Today over 40% of the male patients are in open wards, whilst some months ago a ward of 130 old delusional female patients was made an open ward without the patients causing the slightest trouble.

Open wards have been a feature of Kenmore for some years past. Because I have elaborated a scheme already in operation, no originality is claimed for most of the ideas expressed in this paper. All I do is to claim that these methods are much superior to the old methods for patients, their relatives and the staff.

But the introduction of methods so contrary to accepted teachings requires not only moral courage, but a firm belief in their effectiveness, especially when things are going wrong. It is essential for their successful introduction that one should have sufficient confidence in one's ability to sum up a patient and to prognosticate what he is likely to do in the future. Errors of judgement are inevitable. The nursing staff, too, must be sufficiently versatile to change from the old to the new without becoming unduly worried. Nurse Tamsett, the nurse in charge of the ward, and her assistants were of the greatest possible help to me here. It is also essential that the staff should have sufficient confidence in the administrators of the hospital to know that if they do make an honest mistake, as assuredly they must, they will not be dismissed for it. Insecurity of tenure is one of the major causes of industrial unrest today. If these conditions are fulfilled, I am sure that these experiments, still seriously interfered with by war conditions, will be successful wherever they are tried, and the mental hospitals of the future will be in the same position as the general hospitals of today, trusted implicitly by patients, by their relatives and by the public generally.

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MICRORADIOGRAPHY AND THE TUBERCULOSIS PROBLEM IN AUSTRALIA.

THE diagnosis of tuberculous infection of the lungs has been described in this journal as an old theme. This theme or motif is part of a tuberculosis symphony, as yet unfinished; and though it appears alone, it is present as a contrapuntal part in most of the movements. As a single work it has been presented from time to time with variations; the most complete of these to be presented to readers of this journal during recent months has come from Dr. Reginald Webster, of Melbourne. Dr. Webster has shown that the bacteriological control of radiological findings in the diagnosis of tuberculosis is not only necessary, but can be carried out in the examination of large numbers of men. In the issue of May 10, 1941, we gave an account of a report made by him to the Royal Australasian College of Physicians in which he described the method he adopted in the examination of army recruits. The meeting of the Victorian Branch of the British Medical Association reported in this issue is a natural corollary of Dr. Webster's report—what has been achieved in military circles should if possible be carried into civilian life, and the Victorian Branch discussion makes excellent propaganda to that end.

In view of the work already mentioned, it was natural that Dr. Webster should have been asked to make the opening contribution to the meeting. He gave, as indeed was necessary, a *résumé* of the methods used by him in earlier communications; he also quoted figures comprising his results to June 30, 1941. Dr. Webster's findings in the investigation of four hundred men rejected for military service or deferred because of X-ray evidence of pulmonary tuberculosis show beyond any doubt that if sufficient trouble is taken, active tuberculous infection of the lungs can be unmasked. This is a repetition of a statement made on a previous occasion in these pages, but, even so, it will not, we hope, be a "vain repetition". Anyhow it is the dominant note in our diagnostic theme and should bear repetition. In Dr. Webster's investigations radiological examination of the chest has been the starting point. Readers of this journal should be quite familiar with the value of microradiography in the discovery of tuberculous lesions in the lungs; the contributions of Dr. B. P. Anderson Stuart, Dr. K. E. Shellshear and Dr. Bruce White, of Sydney, published in March and June of this year, and of Dr. H. W. Wunderly, of Adelaide, published in September last year, will probably be fresh in mind. Dr. K. H. Hallam was succinct and also original when he said in the Victorian discussion that microfluororadiography was a speeded-up pectoral policeman who brought culprits to a court of inquiry. We do not propose to discuss in any detail the use of X rays in the diagnosis of pulmonary tuberculosis. To do this would be to traverse the ground already covered by Dr. Hallam in this issue, by the other speakers at the Victorian meeting and by earlier authors in this journal. Two points are obvious. The first is that, though X-ray examination will reveal the presence of a tuberculous lesion, it will not always be possible from the skiagram to determine whether the lesion is active; the second is that in these circumstances the X-ray examination should set in train a series of bacteriological investigations and these would complete the inquiry.

On the last occasion on which microradiography was discussed in these pages a plea was made for the extension to the militia of the routine use of the method which had been exploited with such good effect in the examination of recruits for the Australian Imperial Force. At that time the Minister had refused to accede to the request of the Federal Council of the British Medical Association in Australia that this should be done. Since that time, as a result of further representations made to him, the Minister has reversed his decision. This is a matter for congratulation and marks a forward step in recognition of the need to discover and eradicate tuberculosis from the community. The next step and one that is both logical and obviously necessary is to extend the advantages offered by this method of examination to the civilian members of the community. Already a start has been made in that direction; the following short statement will give some idea of the steps that have been taken. In the first place, for some years a survey of tuberculous infection among medical students has been made in the University of Melbourne by radiological examination and the Mantoux test; for the past two years the radiological examination has been carried out by the 35 millimetre film method (microradiography). This work, which was started as a result of the interest taken by Dr. Eric Cooper and is now carried on by Dr. T. E. Lowe, is really in the nature of an experimental investigation. However, apart from this and from the benefit which the students derive from it, the procedure must have considerable propaganda value, seeing that the subjects are the future medical practitioners of the State. In New South Wales the Department of Public Health has a microradiological service for the members of the community. Tram travellers in Sydney, for example, are confronted by an advertisement stating that early tuberculosis may be seen and not heard, that radiography will reveal an infection before its presence can be detected by the doctor's stethoscope, and that inquiries will be welcomed by the Department of

Public Health. Inquiries and examinations are being made in increasing numbers. In Prahran, Victoria, a microradiological apparatus has been set up in what is known as the Health Centre. X-ray photographs are taken at the cost of twopence per person, not counting overhead expenses, and a survey of young people residing in the city and of those working in the city will shortly be begun. This health centre is part of a "complete central organization for 'total' health" conceived by Dr. R. H. Fetherston, Health Officer of the City of Prahran, and is described in the *Health Bulletin* of the Victorian Department of Public Health for January-June, 1941. The Anti-Tuberculosis Association of New South Wales, a voluntary association affiliated to the National Association for the Prevention of Tuberculosis, London, has recently installed an apparatus for microradiography. The kind of work that may be expected from such an organization is reflected in a statement from the last annual report that of 1,449 contacts of persons suffering from tuberculosis 55 were found to be tuberculous. As far as can be discovered, there are no other centres in the Commonwealth where microradiography is undertaken for civilians—in this discussion we are not considering examination with the five by four inch film, for we believe that for mass surveys the thirty-five millimetre film method is for many reasons the most suitable. In some places the use of microradiography has been attempted or considered. In at least one instance attempts were frustrated by the fact that the necessary apparatus could not be obtained. One of the large hospitals in Sydney is considering the installation of a plant in order to examine the contacts of tuberculous patients. In Western Australia the British Medical Association Branch Council, in the course of negotiations with the University of Western Australia regarding the inauguration of a health service for students, suggested that the Defence Department should be approached in order to discover whether it would be possible to use in connexion with the service the apparatus installed for the examination of recruits. We cannot imagine that this apparatus would be in continuous use by the Defence Department, and in these circumstances it should be possible for arrangements to be made for its use by others for civilian purposes. Suggestions have been made that the proposed health service to students should be extended to include the rest of the civilian population. One last point must be mentioned. In Melbourne it has for at least twelve months been strongly held that women attending antenatal clinics at public hospitals should be submitted to radiological examination to supplement other clinical methods and here microradiography seems to be indicated.

From these statements it is clear that in some places the value of microradiography for mass examinations among the civilian population is realized.[1] There can be nothing but praise for those whose vision and driving power have already been effective. That the use of the method will become universal there is no doubt. Medical practitioners should hasten the day when this will be. They can do it if they will. But we must remember, as already stated, that X-ray examination does not give a result that is the end of the quest—it must be followed by bacteriological investigations of the kind described by Dr. Webster. At the present time far too many sufferers from pulmonary tuberculosis are coming under treatment for the first time when they are in the last stages of the disease. This state of affairs will not be changed until every section of the community is surveyed, and this means mass microradiography.

[1] Melbourne newspapers have recently announced an extension of the practice in that city.

Current Comment.

AN ANALYSIS OF FEMININE FASHIONS.

The day has gone by when Buckle could horrify or amuse his contemporaries by declaring that the total number of suicides or marriages, if a sufficiently large community was investigated, followed certain elementary principles and could with tolerable accuracy be predicted. Since his time many phases of human endeavour, once regarded as determined by individual choice and free will, have been shown to be amenable to statistical analysis and to display, when dealt with in adequate numbers, unmistakable trends which might be described as laws. Nevertheless we learn with a little surprise, not unmixed with mild amusement, that the vagaries of women's dress have been subjected to the same scientific examination. The University of California Press has published amongst its Anthropological Records the results of a laborious research entitled "Three Centuries of Women's Dress Fashions", in which certain fluctuating features of feminine attire, capable of numerical assessment, are treated analytically by Jane Richardson and A. L. Kroeber.[1] Skirt length, skirt width, waist length, waist width, *décolletage* and *décolletage* width have been measured in pictures and fashion plates from early Jacobean times until the late thirties of this century. Put briefly, the main contentions of the investigation are as follows. There appear to be two components in dress fashion; one is mode in the proper sense, that factor which makes this year's clothes different from last year's. These oscillations are, however, small waves superposed on a larger rhythm of style, which shows fair regularity between maxima and minima about fifty years apart, so that the wave length of the periodicity is approximately a century. In addition there are periods of low variability separated by shorter periods of accelerated variability, and the authors endeavour to show that quick changes in dress correspond with times of major wars and unrest. It is not claimed that revolution, wars and social unsettlement directly produce short skirts or extremes of waist, but that they disrupt the established dress style and tend to its replacement or inversion. As is usually the case with statistical inquiries, the causative agents are matters of enthusiastic disagreement. A previous utterance by one of the authors, Professor Kroeber, in 1919, was subjected to considerable criticism.[2] It was claimed that competition or emulation has been the basic force behind dress change; the leaders of society want to surpass the general mass and keep going one step further until a physical limit is reached, and then they turn about and head in the opposite direction. Further, dress can be profoundly influenced by factors which might be called accidental; thus the participation of women in sport led to new departures in clothing and the invention of rayon had repercussions in dress lengths and other attributes of feminine attire. Professor Kroeber in this anthropological publication replies to some of his critics and carries the argument into psychology and the age-old conflict between determinism and free will. We may leave these learned gentlemen to fight the verbal battle to a conclusion if a conclusion is attainable.

Some forty years or so ago W. Ostwald demonstrated the principle that in any mechanism, living or non-living, in which automatic regulation of action is in operation,

[1] "Anthropological Records", University of California, Volume V, Number 2, 1940.

[2] A. L. Kroeber: "On the Principle of Order in Civilization as Exemplified by Changes of Fashion", *American Anthropology*, Volume XXI, 1919, page 235.

the equilibrium established must be oscillatory. When the regulating device or governor is sensitive and works with a minimum of lag, the amplitude of the oscillation is small. That is the rule in the animal body. But if the governor is lacking in responsiveness, then periodicity, or "hunting" in engineering phrase, becomes manifest. In the animal body "hunting" is definitely pathological, as in Cheyne-Stokes breathing; but in human social activities the oscillations are normally of major dimensions; hence in art the florid and the austere alternate over a long time interval, and in conduct the puritanical and the dissolute slowly succeed each other; so we need not be surprised if, in dress, skirts long and short, scanty and voluminous, display the same secular rhythm.

What interests medical men is that the choice of women's dress should be so generally detached from considerations of health. Livers were once compressed until their inhibited functioning produced chlorosis, and in our own day we find high heels causing all sorts of arthritic degenerations in the foot, whilst the vogue of artificial silk has led to quite inadequate protection of the lower limbs from chill in cold weather. The mere male is baffled in his survey of feminine dress and adornment by the fact that natural beauty is so often impaired or actual ugliness engendered. The charm of unpainted cheeks and lips lies in the subtle play of changing tints, and this is completely abolished by rouge and lipstick, which give a mask-like deadness. The gait of a woman with high heels is the very reverse of attractive, as can be plainly seen by comparing it with the unconscious grace of a newspaper boy dodging traffic. How often has a medical student fallen in love with a pretty nurse in uniform, but been sadly disillusioned when he has seen the adored one in mufti with dyed lips and dressed in what she thinks is the correct mode? The most scholarly philologists insist on the absence of logic in the development of human speech; we are forced to assume that a similar syncope of reason dictates feminine styles.

THE TREATMENT OF TETANUS.

The treatment of tetanus is always intensely interesting; apart from the general measures necessary in any grave illness, three essential aims have to be achieved. The first is to control the effects that have already been produced by the toxin on the nervous system, the second is to neutralize the toxin which is passing from the focus of infection to the spinal cord but is still free and has not caused any disorder of nervous function, the third is to limit the absorption of toxin from the infected site.

Tetanus toxin, acting on the motor neurones, causes first muscle rigidity, to which after a variable period are added sudden, exhausting, and cruelly painful spasms. Rigidity alone need cause little concern, but to control the spasms is the urgent and imperative duty of the attending physician. For this purpose sedatives in large doses are usually required and many drugs have been used. Ralph Spaeth, who has recently published two articles on tetanus, strongly recommends "Seconal", a fairly rapidly acting barbiturate.[1] He states that other barbiturates, paraldehyde, morphine, and even inhalation anæsthetics have value, and that each may be indicated in certain circumstances. We must also remember the work that has been done on the use of curare in this disease. Of even greater importance than the choice of sedative is the courage to give adequate doses. There is much truth in the aphorism that enough sedative has been given only when spasms are controlled.

Much thought and work have been directed in recent years toward finding the most rational and effective way to neutralize toxin that has not yet been fixed in the nervous system. As long ago as 1897 Marie produced suggestive evidence that the toxin produced by tetanus bacilli in the infected wound was absorbed by the motor nerve endings in the region of the wound, and passed along the axis cylinders of the peripheral nerves to the spinal cord and brain stem. This view became generally accepted and it was thought that the most effective way to reach and neutralize the toxin was by the intrathecal injection of antitoxin. For a long time many clinicians regarded this as an essential part of the treatment of tetanus. It has a very real disadvantage in that serum injected into the subarachnoid space actually causes an aseptic meningitis, with exudation of many leucocytes into the cerebro-spinal fluid. Cases have been reported in which rapid death followed the administration of tetanus antitoxin by this route. The cause of death seemed to be paralysis of vital bulbar nerve centres, and autopsy revealed a reactionary œdema of the *medulla oblongata* of sufficient degree to result in compression within the *foramen magnum*. Spaeth concludes that to give serum by the intrathecal route in tetanus is both dangerous and irrational. He points out that the serum has no immediate curative effect and no effect on symptoms already established. What successful serum therapy does is to prevent further damage from toxin that has not yet reached the spinal cord. The site of attack on such toxin and the amount of antitoxin required are the subjects of Spaeth's second article.[1] He critically examines the evidence produced by J. J. Abel and other workers in recent years to show that tetanus toxin passes from the focus of infection to the central nervous system by means of the blood stream and not along the peripheral nerves. Many times tetanus toxin has actually been detected in the blood stream of humans or of animals suffering from the disease. The distance of the infected wound from the central nervous system has no apparent effect on the incubation period of the disease, determined in a large series of cases, as one might expect if the toxin spreads slowly, but only slowly, along the peripheral nerves. The frequent occurrence of the earliest evidence of spasticity in a part remote from the wound is one further piece of evidence suggesting a hæmatogenous spread. Spaeth concludes that the spread is mainly, if not entirely, by the blood stream. The obvious way to reach and neutralize this toxin is by injection of antitoxin direct into a vein or into muscles. A number of workers have found that a single dose of antitoxin injected in this way produces a considerable titre of antibody in the blood for several weeks. As a result of repeated titrations of the antitoxin in the blood of his patients Spaeth considers that a single dose of serum is all that need be given. He recommends 120,000 international units for patients treated during the first four days of the illness, and 80,000 international units for those who, less severely affected, come for treatment only after the fourth day.

On the question of surgical removal of the focus of infection to prevent the elaboration of further toxin Spaeth says nothing. It seems an obvious and rational thing to do. Conclusions drawn from single cases are often wrong, but we call to mind one case in which the lesson seems so clear that it cannot be neglected. The patient, a boy, had tetanus and was given sedatives and a large amount of antitoxin by intramuscular injection each day. He had seldom worn shoes, and the soles of his feet bore innumerable puncture wounds from which the infected one could not be selected. It was therefore neglected. The lad slowly improved and after some ten days was considered well enough to need no more serum. Three days later, however, he had again become rigid and was having spasms. Serum was again administered and the two most likely wounds were excised. One of them was found to be a track leading down to a small necrotic pocket deep in the fat and containing tetanus bacilli. The boy then made a rapid recovery. He had been given many times the dose of serum recommended by Spaeth, but it was obviously insufficient to neutralize the toxin being produced continuously in the untreated wound. We are almost inclined to agree that a single dose of antitoxin is sufficient if, and only if, one can be certain that the focus of infection is removed and is not continuing to discharge its lethal product. Much unfortunately remains to be learned of this disease.

[1] *American Journal of Diseases of Children*, July, 1940.

[1] *Ibidem*, June, 1941.

Abstracts from Medical Literature.

PHYSIOLOGY.

The Effects of Anoxæmia on the Pressure of the Cerebro-Spinal Fluid.

FRANK W. MAURER (*The American Journal of Physiology*, May, 1941) reports experiments in which exposure of cats to an atmosphere containing 0·5% of carbon monoxide and to atmospheres containing 6·0% and 8·0% of oxygen respectively, resulted without exception in increased pressure of cerebro-spinal fluid. The average increase during carbon monoxide exposure was 1·74 times the normal. The increases during exposure to 6·0% and 8·0% of oxygen were 1·5 and 1·1 times the normal, respectively. The immediate increases in cerebro-spinal fluid pressure are believed to be due to increased cerebral blood pressure, since the change occurs before the blood oxygen content is effectively lowered. The secondary increase and the prolonged elevation of cerebro-spinal fluid pressure are believed to be due to accumulation of fluid from the cerebral capillaries, whose permeability is increased when the blood oxygen content is effectively lowered. The oxygen content during the secondary pressure increase averages 41%, and at the time of maximum pressure averages 35%.

Peripheral Vascular Responses in Man during Digestion.

D. I. ABRAMSON AND S. M. FIERST (*The American Journal of Physiology*, July, 1941) state that although the influence of the process of digestion on the circulatory system is well recognized, the question arises as to how the blood flow to the periphery is affected. An acceleration of the pulse rate, an increase in pulse pressure and an increase in cardiac output are stated to occur shortly after eating. The method adopted to measure the blood flow has been already described by one of the authors in the *American Heart Journal*, 1939. The venous occlusion plethysmographic method was applied to a series of eight normal subjects. A carbohydrate meal caused no significant changes in the rate of peripheral blood flow in the hand, forearm and leg. At the same time, however, definite increases in the rate of oxygen consumption, in pulse rate and in pulse pressure were observed. After a protein meal there was no change in peripheral circulation for the first half-hour or hour, and then generally the rate of blood flow began to increase, first in the hand and later in the forearm and leg, to remain elevated until the end of the experiment. The changes in oxygen consumption, pulse rate and pulse pressure were similar to those observed when carbohydrate had been taken, except that they were of greater magnitude.

The Storage of Carbohydrate Food.

E. M. MACKAY AND D. R. DRURY (*The American Journal of Physiology*, April, 1941) discuss the form in which ingested carbohydrate is stored in the body. They state that although no one would deny that carbohydrate food in excess of the requirement over relatively long periods was stored as fat, the idea is prevalent throughout the literature on metabolism that oxidation and deposition of glycogen account for practically all of the carbohydrate food ingested under ordinary circumstances. They criticize the previous work of Cori and Cori because their rate of sugar administration was less than half that which might occur under natural conditions. The authors took pairs of female rats, each pair from the one litter, and fed and starved them on alternate days until their food intake on the feeding days and also their weights on the fasting days were constant. The food used was fat-free. These pairs were then killed and liquated in potassium hydroxide solution. The glycogen and fat content of one animal was determined four hours after feeding and of the other at the end of a fasting period. The analytical results indicate that both glycogen and fat were stored, but that the ratio of glycogen increase to fat increase was only 1:10. Under the conditions of the authors' experiment it would appear that ingested carbohydrate was stored mainly as fat and only in a minor degree as carbohydrate. The authors admit that the régime adopted, although necessary for the analytical procedure, is strange to the rat with its usual cycle of feeding during the night and fasting during the day. They feel, however, that even under normal conditions the carbohydrate from the ingested food is stored mainly as fat, only one-fifth being in the form of glycogen. This viewpoint receives further support if the quantitative aspect of carbohydrate ingested to possible glycogen storage capacity is considered.

Age Changes and Sex Differences in Alveolar Carbon Dioxide Tension.

NATHAN W. SCHOCK (*The American Journal of Physiology*, July, 1941) states that alveolar carbon dioxide tension has been shown to be higher in males than in females, and that a corresponding difference in the tension of carbon dioxide in the arterial blood has been demonstrated. He then describes experiments seeking to determine the age at which the sex difference in the alveolar carbon dioxide tension first appears, the extent of the difference and a possible explanation. Fifty normal boys and fifty normal girls (aged eleven to twelve years) were tested at intervals of six months over a period of six years. Two further groups of the same size, in which the children were aged sixteen years, were also examined. The adult groups consisted of twenty male students and fifteen female students, of a mean age of twenty-three years, and an older male group, of a mean age of thirty-two years, and a female group, of a mean age of thirty-three years. The alveolar carbon dioxide tension showed a statistically significant rise in boys between the ages of 11·5 and 17·5 years. No significant change in girls of the same age was noted. The alveolar carbon dioxide tension of adult males is significantly higher than that of adult females. This sex difference first becomes statistically significant at the age of thirteen years. This difference in alveolar carbon dioxide tension of males and females disappears (in adults) if the alveolar carbon dioxide tension for each subject is divided by height or surface area. In persons of one sex no significant correlation between body size and alveolar carbon dioxide tension was found in the present series of observations. A possible explanation for the sex difference must lie in physiological characteristics not present before the age of thirteen years.

Respiratory Modification of the Cardiac Output.

D. H. CAHOON, I. E. MICHAEL AND V. JOHNSON (*The American Journal of Physiology*, July, 1941) describe the results of experiments on dogs in which the direct cardiac volume changes of the two ventricles were measured by a cardiometer during normal breathing. The dogs were under sodium barbital anæsthesia. During inspiration, diminished diastolic size and stroke volume were demonstrated. Systemic arterial pressure also decreased during inspiration, mainly as a result of the diminished stroke volume, since arterial pressure invariably decreased to a greater extent than intrathoracic pressure. During the slow deep respiratory movements following double vagotomy, these effects were exaggerated. Motion pictures were taken of the auricles during normal respiration through plate glass windows sealed into either side of the chest wall. The right auricle became larger in inspiration and smaller in expiration. The left auricle usually was observed to undergo opposite changes, but the changes were never so pronounced as those seen in the right auricle. The decreased size of the left auricle during inspiration indicates that blood is withheld from the left side of the heart during this phase of respiration. It is probable that the increased right auricular size observed in the inspiratory phase indicates that blood is also withheld from the right ventricle as a result of storage in the thin-walled structures on the approach to the right ventricle. Both ventricles must therefore undergo incomplete filling and decreased stroke volumes during inspiration. If, as seems less likely, the greater volume of the right auricle during inspiration indicates greater right ventricular filling, the diminished ventricular volumes must be attributed wholly to incomplete filling of the left ventricle. The right auricle and middle lobe of the right lung were isolated from the circulation, yet left free to expand and collapse with changes in intrathoracic pressure. During inspiration the capacity of both structures increased, indicating a greater storage of blood in both the pulmonary circuit and right auricle during the inspiratory phase.

The Water Intake of an Adult Man with Dysfunctioning Salivary Glands.

F. R. STEGGERDA (*The American Journal of Physiology*, March, 1941) describes some comparative experiments on water intake between normal students and another student who claimed that he had always been without salivary secretions. Examination revealed a complete absence of all salivary glands and ducts. Although the mucous membranes of the mouth were always dry there was evidence of a certain amount of mucinous secretion from gland cells along the inside of the cheeks and under the tongue. These secretions gave no evidence of the presence of amylolytic enzymes and no change in mouth secretion was recorded following subcutaneous injection of six milligrammes of pilo-

carpine. The subject was also deficient in lachrymal secretion, and the early onset of dental caries had left him with complete false dentures at the age of twenty-one years. Contrary to the theory already advanced by Cannon, the absence of salivary glands in man does not cause him to drink greater quantities of fluid than a normal individual. The subject has learned to relieve the dry uncomfortable feeling in his mouth by taking a few swallows of water nearly every hour, although at times he can go without water for two hours. Normal subjects when made thirsty by artificial means, such as exposure of the mouth to a warm dry stream of air or by ingestion of sodium chloride, consume almost 50% more water to moisten the mouth than the subject under experiment habitually drinks. It would appear that a person without salivary glands may have a normal water and salt metabolism.

BIOLOGICAL CHEMISTRY.

Potassium in Animal Nutrition.

E. ORENT-KEIDES AND E. V. MCCOLLUM (*The Journal of Biological Chemistry*, August, 1941) have studied the effects of potassium deficiency on rats. The diets were adequate in all respects, but contained only 0·01% of potassium. The most important outward signs of deficiency were roughness and thinning of the fur, a striking alertness and a pronounced pica. Growth occurred at a slower rate than normal, but there was at no time a loss of weight, and longevity appeared unaffected. Œstrus rhythm in rats on the low potassium diet was disturbed; ovulation was irregular and at a slower rate, while sexual maturity was delayed. In some animals œstrus was entirely suppressed. In males the spermatozoa were defective in number, form and motility. In spite of the low potassium intake, the deficient rats remained in equilibrium with respect to this element; storage of magnesium was 2·5 times greater than in control rats; chlorine balance, on the other hand, was negative. Tissue analyses showed that when the potassium intake was low the potassium content of muscle, heart and kidney was lowered. It was evident that the animal organism possessed a remarkable capacity to withstand potassium deprivation for long periods of time in spite of obvious injury to heart and kidney.

Determination of Plasma Uric Acid Content.

H. A. BULGER AND H. E. JOHNS (*The Journal of Biological Chemistry*, August, 1941) have questioned the reliability of methods now in use for the determination of the blood uric acid level and have therefore attempted to estimate the true uric acid content of plasma. This was accomplished by determination of the reduction, by protein-free filtrates, of an alkaline ferricyanide solution, under special conditions, before and after the destruction of uric acid by uricase. As a result of the studies defects in certain methods and probable explanations for many discrepancies were disclosed. A large and variable positive error may be introduced by the action of non-uric-acid reducing substances, especially in whole blood. A negative error may result from the quantity of uric acid which is lost during the precipitation of protein from whole blood. A large negative error is introduced by the inhibition of colour development in colorimetric procedures, by certain factors in protein-free blood filtrates. The true uric acid content of plasma was found in normal subjects to lie between 2·0 and 6·0 milligrammes per 100 cubic centimetres. Attempts to apply the method to whole blood proved unsuccessful.

Iodine Metabolism.

M. E. MORTON, I. PERLMAN AND I. L. CHAIKOFF (*The Journal of Biological Chemistry*, August, 1941) have investigated the effect of thyreotropic hormone on the rate at which labelled thyroxine and di-iodo-tyrosine appeared in the thyreoid and plasma following the injection of radioactive (labelled) iodine into guinea-pigs. The effects on guinea-pigs treated with thyreotropic hormone were compared with those on normal animals. At both time intervals studied (2 and 26 hours after injection) a larger part of the radioactive iodine was bound organically by the hyperactive than by the normal gland, and similarly, the plasma of hormone-treated animals contained larger amounts of organically bound labelled iodine than did plasma of control animals. In the thyreoid glands a larger proportion of labelled iodine was always present as di-iodo-tyrosine than as thyroxine; but in the hyperactive glands the relative proportion as thyroxine was greater than that in normal glands. In the plasma at two hours a larger proportion of the labelled iodine was present as di-iodo-tyrosine than as thyroxine, while at 26 hours the reverse occurred. In hormone-treated animals the plasma at 26 hours contained 80% of the labelled iodine as thyroxine, as compared with 50% in the plasma of normal animals. It was evident that despite the fact that the thyreoid gland of treated animals had been depleted of its iodine and thyroxine under the influence of the thyreotropic hormone, the capacity of the hyperactive gland for removing recently injected iodine and converting it to organic compounds was increased greatly above normal.

Skeletal Changes in Vitamin A Deficiency.

E. MELLANBY (*The Journal of Physiology*, June, 1941) has studied the effect of vitamin *A* deficiency on the development of skeletal abnormality in dogs and the relation of this to changes in the nervous system. It is shown that one of the functions of vitamin *A* is to influence the structure of growing bone, probably by limiting the number and the degree of activity of osteoblasts and osteoclasts. In animals suffering from a deficiency of vitamin *A* osteoblastic and osteoclastic activity is increased, resulting in proliferation of cancellous at the expense of compact bone, and causing many bones to lose their normally fine moulding and outline and to become thickened and enlarged. The position of some of the bone overgrowths in the skull and vertebral column results in degenerative changes in the brain and in cranial and peripheral nerves. Overgrowth of the cranial bones may press on and produce deformity of parts of the brain. The greatest hypertrophy is found in the bones forming the posterior fossa of the skull, so that the *medulla oblongata*, pons, cerebellum and nerves in close association with these parts of the brain are more particularly affected. Most of the cranial nerves of young dogs are liable to be compressed and to suffer degenerative changes if the defective diets are continued over long enough periods. The posterior root ganglia and the anterior root nerves of the spinal cord may also be squeezed and the nerve fibres destroyed by overgrowth of the vertebral bones. In advanced cases of deficiency there is a substantial increase in intracranial pressure. The excessive activity of the osteoblasts and osteoclasts is compared with the well-known hyperplasia of epithelial cells in vitamin *A* deficiency.

Alcohol of Pulmonary Air.

H. W. HAGGARD, L. A. GREENBERG, D. P. MILLER AND R. P. CARROLL (*The Journal of Laboratory and Clinical Medicine*, June, 1941) have conducted a careful investigation of the method of estimation of alcohol in pulmonary air as employed for the indirect determination of the concentration of alcohol in the blood. It was shown that the coefficient of distribution of alcohol between air and blood as previously used, 1:2,000, was too high and the correct value as determined both *in vivo* and *in vitro* was 1:1,300. The discrepancy was in part due to the loss of alcohol in condensed water vapour in the containers used to collect expired air. Further, the concentration of alcohol in the pulmonary air could not be correctly calculated from the concentration in mixed expired air on the basis of carbon dioxide content; this was because alcohol diffused more rapidly into the air of the respiratory dead space than did carbon dioxide. The concentration of alcohol in alveolar air was found to correspond with that of "venous" air obtained by a short period of rebreathing, in both trained and untrained subjects. A method is described in detail for obtaining "venous" and alveolar air without loss of alcohol. The procedure permits an accurate determination of concentration of alcohol in blood from analysis of that in pulmonary air. The errors in methods previously used in medico-legal investigations are discussed in detail.

The Blood Iodine Content in Thyreoid Disease.

D. S. RIGGS, E. F. GILDEA, E. B. MAN AND J. P. PETERS (*The Journal of Clinical Investigation*, July, 1941) have reinvestigated the level of iodine in the blood of patients with thyreoid disease, because it had been found that many of the older methods of analysis yielded erroneously high values. In 26 normal subjects the range of values was 2·4γ to 4·2γ iodine per 100 cubic centimetres. Seven patients with myxœdema had iodine levels from 0·3γ to 1·7γ *per centum*. Thirty-one patients with clinical evidence of hyperthyreoidism had iodine levels from 6·4γ to 21·9γ *per centum*. It was evident that elevated blood iodine values were always found in hyperthyreoid patients; but on the other hand, raised iodine levels were found in some patients without manifest hyperthyreoidism. In these patients the symptoms appeared to be more closely related to disorders of the nervous system than thyreoid over-activity. A group of six patients with nodular goitre, but without symptoms of hyperthyreoidism, had blood iodine values that did not differ significantly from the normal.

British Medical Association News.

SCIENTIFIC.

A meeting of the Victorian Branch of the British Medical Association was held on August 6, 1941, at the Medical Society Hall, East Melbourne, Dr. H. Boyd Graham, D.S.O., M.C., the Acting President, in the chair. The meeting took the form of a symposium on the diagnosis of pulmonary tuberculosis.

Modern Methods in the Diagnosis of Tuberculosis.

Dr. Reginald Webster discussed the diagnosis of pulmonary tuberculosis with special reference to bacteriological control of radiological diagnosis. He said that the diagnosis of pulmonary tuberculosis was now a laboratory matter, a function of the X-ray laboratory, complemented by the bacteriological laboratory.

The most carefully and competently applied clinical methods often failed in the detection of incipient and even advanced tuberculous disease of the lungs, and intensive and world-wide radiological study had shown the futility of the vague and elusive auscultatory signs upon which diagnosis formerly rested. But the interpretation of a thoracic radiograph was after all a matter of opinion; in many films the diagnosis of pulmonary tuberculosis seemed to be writ large, but there were many others in which experienced radiologists preferred to reserve judgement. That so much discussion had centred upon the question of the radiological estimation of activity was evidence of the limitations of radiology in that important phase of diagnosis. Clinicians should never forget, in their natural eagerness to render due credit to radiology and to avail themselves of its indispensable aid, that the ultimate and only unequivocal proof of the existence of tuberculous disease—pulmonary, osseous or genito-urinary—was the demonstration of the tubercle bacillus.

The diagnosis of pulmonary tuberculosis was fraught with so much anxiety to the patient, such concern for his health and fear of economic and social disability, that it was only fair to him that when such a diagnosis was based upon radiological evidence it should be certified, audited and found correct, if possible, in the bacteriological laboratory. Dr. Webster did not think it necessary for him to multiply reasons why bacteriological confirmation of radiological diagnosis of pulmonary tuberculosis was essential, and he therefore proceeded to discuss what he considered should be a minimum bacteriological routine.

One of the first exercises given to students ever since a course of bacteriology found a place in the medical curriculum was to prepare and stain a film of sputum by the Ziehl-Neelsen method, and it was a remarkable and regrettable fact that ever since Koch's discovery of the tubercle bacillus in 1882 until recent times this elementary and inadequate procedure had remained the standard for bacteriological diagnosis and control of pulmonary tuberculosis. By that clinical colloquialism, the term "positive sputum", it was generally to be understood that acid-fast bacilli were in evidence by the microscopic search of smear preparations; its opposite number, "negative sputum", was obviously a relative term, the significance of which depended entirely upon the efficacy of the methods employed to detect tubercle bacilli and the tenacity with which such methods were pursued. A "negative" report issued after a fifteen-minute search for acid-fast bacilli in a direct smear preparation of sputum, even though a number of specimens were so examined, was clearly not of the same value as a similar finding based on the collection of the total output of sputum of any given patient during a period of seven days, its digestion, concentration and subsequent subjection to cultivation by multiple sowings on a variety of appropriate media—an exhaustive procedure which had been put into practice by H. S. Willis and R. G. Kelly at the Maybury Sanatorium, Michigan, in the United States of America.

In discussing laboratory methods in the bacteriological diagnosis and control of pulmonary tuberculosis, Dr. Webster emphasized that, as a means of sifting sputum and other tuberculous exudates for tubercle bacilli, a sieve of very coarse mesh was used. It had been computed by H. J. Corper that tubercle bacilli were not detectable in smears until their numbers approximated 100,000 per cubic centimetre, whereas guinea-pig inoculation and proved methods of cultivation would disclose them in numbers as low as 10 to 100 per cubic centimetre. The much-quoted figure of Corper had been regarded as unduly pessimistic by Bogen and Bennett, who, basing their calculation upon experience in Gaffky counts and the assumption that a careful worker would examine at least 300 microscopic fields before issuing a "negative" report, estimated that the presence of 1,000 microorganisms per cubic centimetre of sputum should permit of the discovery of tubercle bacilli by direct microscopic examination. Wherever the truth might lie between these two standards, it was certain that cultivation frequently revealed the presence of tubercle bacilli even when repeated, patient and conscientious search of film preparations left no alternative but to record a negative finding. The technique of culture of *Mycobacterium tuberculosis* had now advanced to such a stage that many workers asserted that the cultural method equalled in sensitivity and reliability the time-honoured practice of guinea-pig inoculation. That this belief was well founded might be judged from the work of Shrewsbury and Barson, of the University of Birmingham, who showed that the method of culture employed by them yielded 100% of successful cultivations of the tubercle bacillus from a diversity of specimens embracing sputum, cerebro-spinal fluid, urine, fæces and pus. In a recent paper in The Medical Journal of Australia Dr. Webster had recorded his own results in the cultivation of *Mycobacterium tuberculosis* as 100% successful in 65 samples of cerebro-spinal fluid, 96% successful in 26 tuberculous bronchial glands, 90% successful in morbid material derived from 72 tuberculous bones and joints, and 66% successful in 36 specimens of tuberculous cervical glands. There were special reasons operating for the relatively low figure in connexion with tuberculous cervical adenitis; he had discussed them in the paper to which he had referred.

The work of Shrewsbury and Barson, as it referred specifically to sputum, showed that of 135 samples of sputum in which tubercle bacilli could not be detected microscopically, 29 (21·4%) were found to contain tubercle bacilli by cultural examination. Similar findings had been recorded by C. A. Green, from the bacteriological department of the University of Edinburgh. Green reported that in the examination of 2,796 specimens of sputum for the presence of tubercle bacilli, the positive findings by cultivation exceeded by nearly one-third (32·4%) those attending the microscopic examination of films. More striking perhaps, in this particular, were the figures of Corper and Cohn, who had published results of a routine examination for tubercle bacilli of specimens of sputum in which acid-fast bacilli could not be found by microscopic search. Of 100 samples of sputum, in which negative results had been consistently obtained in regular monthly examinations of film preparations over periods ranging from four months to two years, no less than 50 yielded a positive finding by a single cultural test.

Such findings illuminated the loop-holes in the film method and constituted a compelling indication for the adoption of cultural methods as a routine procedure in the bacteriological examination of specimens of sputum, with regard to which clinical suspicion of pulmonary tuberculosis could not be confirmed by direct microscopic examination.

Dr. Webster went on to say that shortly after the institution of routine thoracic radiography in the medical examination of volunteers for enlistment in the Australian Imperial Force, he undertook the bacteriological examination of recruits rejected or deferred by reason of radiological evidence of pulmonary tuberculosis. Both the magnitude of the task and the necessity for working within army requirements for prompt disposal of the men precluded, in the great majority of instances, more than one attempt to establish the diagnosis on a bacteriological basis. It was evident that the microscopic search of films would not meet the situation; if there were to be one examination only, it was essential that it should be as searching as possible. The time at his disposal did not allow of discussion of the relative merits of the cultural method and guinea-pig inoculation. He was convinced that there was little between the two, and for various reasons preferred direct cultivation to animal inoculation for the detection of microscopically elusive tubercle bacilli. As a means of coping with large numbers of specimens, such as were in prospect and soon materialized from Australian Imperial Force recruits, guinea-pig inoculation presented itself to him as slow, cumbersome, expensive and totally impracticable. Hence direct cultivation of *Mycobacterium tuberculosis* became at the same time the method of choice and the dictate of necessity.

In planning the large-scale bacteriological examination of recruits Dr. Webster had anticipated that many of these apparently healthy young men would be unable to produce any sputum; it was therefore arranged that the men should report at the laboratory at 9 a.m., having had no food or drink since the previous evening. In the event of a man's being unable to produce expectoration that could be approved as genuine bronchial mucus, the opportunity was taken to withdraw a sample of the content of the fasting stomach by means of the Rehfuss tube. It had been remarkable how well the men had cooperated in the ceremony known among

themselves as "swallowing the snake". In introducing the subject of examination of the gastric content, Dr. Webster said that the demonstration of tubercle bacilli in the mucus aspirated from the fasting stomach had long been familiar in pædiatric practice, forty-one years having elapsed since Meunier first introduced the method to overcome the difficulty occasioned by the fact that children invariably swallowed sputum rather than expectorated it. Meunier's work did not attract the attention which it undoubtedly merited until it was applied by Armand Delille and Vibert in 1927. Since that year a number of workers in the field of pædiatrics, notably Wallgren in Sweden, Poulsen in Denmark, and Collis and Brockington in England, had established the method as one of very great clinical value.

Influenced by the fact that many specimens of so-called sputum did not represent mucus of bronchial origin, and by the further consideration that many persons excreted small amounts of bronchial mucus which they swallowed rather than expectorated, a number of Continental workers, led by Armand Delille, Lestocquoy and Vibert (1930), had applied Meunier's principle to the examination of adults. They had been followed by Kane and Hounslow in England, whose work represented the only attempt so far recorded on the part of English workers to extend to adult practice a technique which had yielded such striking results among children. Kane and Hounslow had reported findings in which, by cultural and animal inoculation methods, tubercle bacilli were shown to be present in the gastric content of 17 out of 55 sanatorium patients who either produced no sputum or provided specimens in which both direct and cultural methods of examination failed to detect tubercle bacilli.

A convincing demonstration of the importance of directing attention to the content of the fasting stomach in the endeavour to obtain bacteriological confirmation of radiological indications of pulmonary tuberculosis had been provided by the work of R. H. Stiehm, Director of the Department of Student Health in the University of Wisconsin. Stiehm assembled 50 students, radiological examination of whom denoted the presence of what he termed "minimal" pulmonary lesions. In none of the group was there any clinical evidence of tuberculosis; all the students regarded themselves as well, and in such samples of sputum as they could produce the tubercle bacillus could not be demonstrated. The next step was to subject each student to aspiration of the content of the fasting stomach on each of three successive mornings, in order to examine the product for the presence of tubercle bacilli by microscopic search and animal inoculation. Acid-fast bacilli were found in films of the gastric mucus of nine of the 50 students comprising the group, but in no less than 36 (72%) tubercle bacilli were revealed in the gastric content by guinea-pig inoculation. In 36 instances, therefore, the radiological findings were endorsed and the presence of an unhealed lesion was unequivocally demonstrated.

Dr. Webster suggested that the best manner in which he could illustrate the operation of the methods which he had outlined was to give some account of his work in connexion with the radiological survey of the Australian Imperial Force. The time consideration obliged him to restrict discussion of the criteria observed for the identification of *Mycobacterium tuberculosis* to a brief formulation. Acid-fast bacilli discovered by direct microscopic examination of smears of sputum, or cultivated from the sputum of men whose lesions were assessed radiologically as "active", were accepted as *Mycobacterium tuberculosis*. In every case in which an acid-fast bacillus was recovered by culture from the gastric mucus, irrespective of the character of the radiological report, the virulence of the culture for the guinea-pig was determined. Similarly, acid-fast bacilli cultivated from the sputum of men with regard to whom X-ray findings were interpreted as indicative of old, healed, inactive or doubtful tuberculosis were required to prove their capacity to induce fatal tuberculosis in the guinea-pig. In both groups an interim report of "strong presumption" of tuberculosis was issued pending the result of the animal inoculation test.

The number of men for whom examinations were complete at the end of June, 1941, was 461. Of these 61 were referred in error; their skiagrams had been questioned, but on grounds other than that of suspected tuberculosis. There remained, therefore, 400 men rejected or deferred by reason of X-ray evidence of pulmonary tuberculosis, bacteriological examination of whom was undertaken. The number shown to be actively discharging tubercle bacilli was 129, or 32·2%.

The methods by which the positive bacteriological findings had been determined in the 129 cases were the following: direct smear examination of sputum, 41; by cultivation from sputum in which tubercle bacilli could not be demonstrated by microscopic search, 61; by culture from the gastric mucus of men from whom no sputum was obtainable, 27. The application of cultural methods and the utilization of gastric contents in the absence of sputum had therefore resulted in 129 positive bacteriological findings, as against 41, which would have been the number had the examinations been restricted to smears of sputum. The average reporting time for sputum cultures was 18·6 days, and the average for gastric mucus cultures 21·6 days. That the figure for positive results obtained by the microscopic examination of smears of sputum (41) compared so unfavourably with that attending sputum cultures (61) could not be attributed to hurried or inadequate scrutiny. Considerations of economy of effort and culture medium, conservation of incubator space, and the possibility of summary disposal of the specimen with an immediate positive report, were strong inducements to persevere in the search for acid-fast bacilli in the films. The positive findings attending cultivation from the gastric mucus (27) had all been verified by virulence tests and proof of the ability of the cultures to induce progressive, widespread and fatal tuberculosis in guinea-pigs. It was of interest to note that in eight men acid-fast bacilli, subsequently shown by cultivation and guinea-pig inoculation to be virulent tubercle bacilli, were detected by the microscopic search of direct smears of the gastric mucus.

Dr. Webster said that seven instances of cultivation of acid-fast bacilli from the gastric content had not been included in his statement of results. Three were omitted because the men who provided them had already been included in the group whose condition was confirmed bacteriologically by the detection of tubercle bacilli in film preparations of sputum. Of the remaining four, in two the bacilli were non-virulent to guinea-pigs, and in two the virulence remained undetermined, owing to the late appearance of contaminants in cultures which were perforce neglected for some time owing to pressure of work.

Of the 129 men concerning whom bacteriological proof of active tuberculosis had been adduced, 74 continued to give negative answers to specific questions regarding hæmoptysis, pleurisy, sanatorium treatment and the occurrence of pulmonary tuberculosis among their near relatives. A few spoke lightly of a "cigarette cough", while many declared that, childish illnesses excluded, they had never had occasion to consult a doctor and had never lost a day's work. Affirmative replies in respect to one or more points in the cross-examination which Dr. Webster had indicated were received from the remaining 55 men. Nine had experienced hæmoptysis and 21 had suffered an attack of genuine pleurisy. Two were obviously ill when they were presented for bacteriological examination. Ten had formerly been inmates of sanatoria, and 23 confessed to the occurrence of pulmonary tuberculosis in their own families or among their near relatives. Such information was no doubt suppressed by these men at the time of their enlistment and preliminary medical examination; but with the realization that they had been weighed in the radiological balance and found wanting, they evidently felt that they could gain nothing by further concealing the facts of their clinical histories.

In proceeding to demonstrate by means of lantern slides the correlation of radiological and bacteriological findings, Dr. Webster intimated that the radiological reports upon which such correlation was based were those issued as interpreting the 14-inch by 17-inch films, and not those of the miniature screen photographs. Group I consisted of 200 men whose skiagrams were regarded as indicative of active pulmonary tuberculosis; the number of positive bacteriological findings was 108, or 54·0%. Group II consisted of 171 men radiologically designated as subjects of old, healed, inactive or doubtful tuberculosis; there were 14 positive bacteriological results, or 8·2%. *Mycobacterium tuberculosis* was recovered by culture from the sputum of nine of the men in Group II and by gastric mucus culture from three; and in the remaining two instances acid-fast bacilli were found in Ziehl-Neelsen preparations of the sputum. All nine sputum cultures, as also the three gastric mucus cultures, induced severe and fatal tuberculous lesions in guinea-pigs.

Dr. Webster indicated that in a report which he had prepared for the Director-General of Medical Services he had included a table assembling the clinical, radiological and bacteriological data relating to the men in this interesting group. As the table was rather long and contained too much detail to show in a lantern slide, he had selected the findings pertaining to three men, whom he presented as typical of the group (Table I).

In the case of number 37 it was ascertained that this soldier had experienced some pain in the chest fifteen months previously; on that occasion he was not obliged to go to bed, and he had otherwise always been well. Number 167 was a man who returned to his home in Sydney after his discharge from the army in Melbourne. He presented himself at the Anti-Tuberculosis Clinic, Surry Hills, where

TABLE I.

Examples from Group II.

Serial Number.	X-Ray Report.	Bacteriological Report.
37	Scattered calcification, right apex.	Positive gastric culture.
167	Small calcified nodules in apex of left upper lobe. No evidence of activity and remainder of lungs normal.	Positive sputum culture.
225	Small calcified nodules at left apex. No definite evidence of activity.	Positive sputum culture.

a further skiagram was taken. So little of diagnostic significance was apparent on the film secured in Sydney that Dr. Bruce White caused a letter to be addressed to Dr. Webster, conveying a request for any information he could give concerning this man. Dr. Webster had replied that he had cultivated an acid-fast bacillus with all the cultural characters of *Mycobacterium tuberculosis* from the sputum, and would proceed with guinea-pig inoculation. The result of the animal inoculation test was conclusive. Number 225 stated that he had suffered an attack of "dry" pleurisy fifteen years earlier; he was not sufficiently ill to be confined to bed at that time. He could not remember any other illness.

Dr. Webster urged that, although the figure for the proportion of bacteriological findings was high, particularly in Group I, in which it reached 54% among 200 men designated on radiological evidence subjects of active pulmonary tuberculosis, it was of necessity an under-estimate. As had been indicated, the men passed the laboratory way but once, and it was only in exceptional instances that repeated examinations had been feasible. Were it possible again to comb through those men for whom a negative bacteriological finding had been recorded, it was more than probable that the tubercle bacillus would be uncovered in some of them.

There remained three men, to be placed in a third group, in view of the fact that no X-ray evidence of pulmonary tuberculosis was apparent. Dr. Webster had prepared a lantern slide relating to this group (Table II).

TABLE II.

Group III.

Serial Number.	X-Ray Report.	Bacteriological Report.
238	Lungs clear: Pass.	Positive sputum culture.
253	Pass.	Positive sputum culture.
255	No sign of tuberculosis: Pass.	Positive gastric culture.

That the cultures from these three men were those of genuine and virulent tubercle bacilli was proved by guinea-pig inoculation in each instance.

It was stated by number 238 that his father had died of pulmonary tuberculosis one year earlier; there were no suggestive facts in the personal and family history of number 255. In the case of number 253, it was ascertained that he had been sent for examination in mistake for his brother, who had been summarily rejected by the radiologists. Number 253, however, gave a history of pleurisy only four months before he enlisted, and from a second sample of sputum acid-fast bacilli were again cultivated. That number 238 and number 255 presented themselves for bacteriological examination must have been fortuitous in view of the X-ray reports with regard to them, and three such unanticipated bacteriological results were startling and provocative of reflection.

Dr. Webster went on to say that to the three men in Group III was to be added another, the facts concerning whom came to his knowledge in the course of laboratory work which he had been discharging for Number 2 Royal Australian Air Force Hospital. On January 31, 1941, a radiological report of "infiltration in left mid-lung" was issued with regard to a certain officer; a comment was added that the condition was probably non-tuberculous and a suggestion that the examination be repeated after the lapse of a few weeks. A second skiagram was taken on February 28, the report on which stated that there was then no definitely abnormal feature to be observed in the lungs. After a third X-ray examination on April 2 a ruling was given that the condition had resolved and that there was no radiological evidence of tuberculosis. In the meantime, unaware of the clinical course and radiological findings, Dr. Webster had recovered an acid-fast bacillus with all the cultural characters of *Mycobacterium tuberculosis* from this officer's scanty sputum. The identity of the microorganism was confirmed by guinea-pig inoculation, and a few weeks later *Mycobacterium tuberculosis* was cultivated from a second sample of sputum.

Dr. Webster said that the findings which he had recorded for these four men were not unique, and those present needed to search no further than their sister capital cities in Australia for similar observations. In Sydney recently Dr. Bruce White had stated, in a paper presented to the New South Wales Branch of the British Medical Association, that during the last few years he had gained knowledge of at least six patients whose original skiagrams showed no abnormal shadows, despite the presence of tubercle bacilli in the sputum. On a similar occasion in Adelaide, in September, 1940, Dr. Darcy Cowan cited the case of a patient in whose sputum tubercle bacilli were readily demonstrable at the time of issue of a radiological report of "healed and calcified lesion at the left apex". Dr. Darcy Cowan's patient would take a place with those Dr. Webster had presented as Group II, and the cases mentioned by Dr. Bruce White fell into Group III. An expression of opinion by one or more radiologists might evoke some profitable discussion. Dr. E. Sydney Morris had advanced the suggestion at the last annual meeting in Sydney of the Royal Australasian College of Physicians that the findings which distinguished the men in Group III were explicable on the basis of "tracheo-bronchial" or "bronchoscopic" tuberculosis.

In conclusion, Dr. Webster remarked that not without trepidation he approached the contentious question of the radiological estimation of activity of a tuberculous lesion, assuming it to be within his province and capacity. He was prepared to concede that it might exceed his capacity, but did not consider that it lay beyond his province. As a pathologist he was of opinion that a statement to the effect that a tuberculous pulmonary focus was healed was one that could never be made with certainty, by physician, radiologist or pathologist. Many researches had shown that a high proportion of apparently old, dry, caseous and calcified pulmonary foci and lymphatic glands harboured viable and virulent tubercle bacilli. Perhaps the most notable of such researches was that of Opie and Aronson, and in this connexion the name of Opie commanded immediate attention. In an exhaustive study of latent, unsuspected, and even apparently healed tuberculous foci in the lungs and tracheo-bronchial glands of 169 subjects who had died from causes other than pulmonary tuberculosis, Opie and Aronson demonstrated that the material derived from such foci induced tuberculosis in guinea-pigs in 30% of the tissues examined. Among the lesions investigated were fibrous apical scars, to all appearance soundly healed, and indeed showing no microscopic evidence of persisting tuberculosis; approximately one-fourth of such scars were proved to contain living tubercle bacilli. Fibro-caseous lesions of the pulmonary apex, unsuspected clinically and discovered incidentally in routine autopsy work, in most instances accommodated virulent tubercle bacilli. If, therefore, the pathologist, in his position of advantage with the dissected specimen and microscopic sections at his disposal, could not determine healing without recourse to bacteriological methods, it was obvious that such a decision could not be reached on radiological grounds. Dr. Webster suggested that frankly exudative lesions and cavitation, as observed radiologically, might properly be regarded as evidence of activity; that fibrosis and calcification might fairly be interpreted as indications of a tendency to limitation and retrogression of the pathological process; but that positive and emphatic statements regarding healing and activity were scarcely justified.

DR. KEITH HALLAM discussed the use of X rays in the diagnosis of pulmonary tuberculosis. He said that like many other therapeutic or diagnostic agents, X rays had been glamorized and the resultant tendency had been to overestimate their value—so much so that they had in many cases become the arbiter of the fates of patients who were sent along to have their lungs radiologically examined. But the physician who used the X rays as a specialty did not pose as a kind of radiological Judge Jeffreys, butchering the destinies of patients who showed a few shady patches in their lungs, for these shady patches might not be due to tuberculosis. The radiologist stood forth rather as a witness to give his evidence—evidence to be sifted and judged on its merits. Radiology had played varying parts in the

diagnosis of pulmonary tuberculosis, and these might be assigned roughly to three eras. At first radiography of the lungs was so poor that only the grossest lesions were demonstrable. Then came the period when technique almost suddenly became so good that pulmonary radiographs were fanatically worshipped as a divine revelation of pathological changes. At the present time they were attempting to assess, sanely and without prejudice, the correct value of the X-ray film as an aid to the diagnosis of pulmonary tuberculosis. Some few indeed remained literally in the dark ages by trying to detect early lesions from fluoroscopic examinations alone, or from shockingly bad films; others pathetically placed great reliance on the film to the exclusion of other evidence. In Dr. Hallam's experience many persons had been sent to the Tuberculosis Bureau with recommendations for admittance to sanatoria on the evidence only of radiographs taken by those inexperienced in the interpretation of pulmonary skiagrams. After investigation at the bureau had proved such persons not to be suffering from tuberculosis, they had to be sent home, at times as far as two hundred miles.

Dr. Hallam went on to say that there was no shadow of doubt that in almost every case of suspected pulmonary tuberculosis discussion had to centre round the X-ray appearances, and their value would be in a descending order to three groups of medical practitioners: (i) those who consulted with the radiologists; these were the answer to a radiologist's prayer; (ii) those who implicitly relied on the X-ray report and expected the radiologist to make the diagnosis; this was flattering, but unwise; (iii) those who ignored the radiologists and trusted to their own judgement to interpret what they saw on the films. The patients of these physicians got a "poor deal", as the personal contact of the physician and radiologist engendered an interchange of knowledge and a mutual understanding of the patients under discussion. The value of X-ray films to those willing to accept them at their true value might be sorted into four grades: (i) Cases in which one might reasonably, without other evidence, state that the X-ray appearances were due to tuberculosis. These cases were rare in private practice. In mass surveys of recruits in which, before being radiologically examined, the men were passed as fit by medical officers, X-ray evidence of tuberculosis might be regarded as conclusive. (ii) Cases in which the history and symptoms, associated with other evidence, made one certain that the pathological change revealed by the film was tuberculosis. (iii) Cases in which the X-ray appearances were equivocal and the diagnosis was in the balance till some positive evidence from another source was forthcoming. This might be from the sputum examinations or from serial X-ray examinations. (iv) Cases in which the X-ray appearances were misleading. In this category were those cases in which the X-ray findings were negative, but in which tubercle bacilli were found in the sputum. This occurred in bronchial tuberculosis. The pathological changes caused by pulmonary tuberculosis were protean. There might be increased vascular markings, areas of haziness, nodules, cavities, fibrosis, calcifications and areas abnormally radiolucent. All these pathological changes might be caused by some agent other than Koch's bacillus.

Dr. Hallam went on to discuss the differential diagnosis. He said that one frequently read of or came across in practice many conditions which might be confused with pulmonary tuberculosis, and he gave three quotations from *The American Year Book of Radiology* (1940), which he described as a compendium of extraordinarily high quality. (i) "Roentgen findings in all bronchomycoses simulate pulmonary tuberculosis." (ii) Concerning Boeck's sarcoid: "In a certain number of cases the pulmonary changes could hardly be distinguished from ordinary chronic tuberculosis of the predominantly fibrotic type." (iii) "The appearance of the chest [in a case of coccidiosis] when compared with that of another patient in whom miliary tuberculosis was proved, provided no distinctive criteria on which to base an accurate differential radiological diagnosis." Other conditions which might be confused with pulmonary tuberculosis were: disseminated focal pneumonia, pseudo-tuberculosis of the lungs associated with anæmia, Friedländer's lung infection, post-influenzal lung appearances, cardiac conditions, malignant conditions, syphilis, abscesses and cysts, silicosis and several others.

It was possible to distinguish between activity and inactivity only after viewing at least two films taken at an interval of several weeks.[1] Collateral X-ray evidence was of the greatest importance; signs of fibrosis, of calcified nodules in the lungs and of old pleuritic infection were valuable evidence when one was attempting to determine whether a patient was suffering from a current tuberculous lesion. If the patient had tuberculosis in some other part of the body, suspicious shadows in the lung fields impressed one as being probably tuberculous in origin.

Dr. Hallam then referred to modern methods of examination, first discussing tomograms. He said that by this method of examination only thin layers of the patient were in focus, and shadows of all tissues anterior and dorsal to this layer were diffused by the movements of the apparatus. One result was that cavities in the lung could be precisely visualized and located, in spite of overlying or underlying fibrotic lung or pleura. A tomographic examination was of inestimable value in the primary detection of cavities, and in what might be designated the secondary detection of cavities; by this Dr. Hallam meant the decision whether cavities were still present after artificial compression of the lung by pneumothorax or thoracoplasty. There was no need for him to dilate on the importance to the physician and surgeon of knowing whether the object of their treatment (obliteration of cavities) had been achieved.

Speaking of microfluororadiography, Dr. Hallam said that De Abreu, of South America, had made the first large-scale surveys by this method in 1936. A debt was owed to Major Douglas Galbraith, of whose report on tuberculosis in the United States of America a special abstract had been published in THE MEDICAL JOURNAL OF AUSTRALIA of February 22, 1941. From a close study of this, one was able to acquire a background for viewing the panorama of tuberculous manifestations seen from day to day. Microfluororadiography was the universal method of mass radiography. It lent itself to this purpose because of its cheapness, the speed with which a large number of subjects could be examined, and the rapidity with which the photographs could be viewed and reported on. Briefly, the method was to photograph the screen images of the chest on reels of 35-millimetre films, the shutter coordinating with the X-ray fluoroscopic flashes. A reel of 36 exposures could with good organization be made in a quarter of an hour; that was a rate of about 150 an hour, or 1,500 in a working day of ten hours. This small film, when projected onto a screen to a size of approximately 15 inches by 12 inches, gave a good impression of the condition of the thoracic contents; but when there was any doubt as to the significance of certain shadows, an ordinary 14-inch by 17-inch film was necessary to ascertain minute details. As radiography by means of the 14-inch by 17-inch film method was more accurate than microfluororadiography, champions of the former said that it should invariably be used. This was an ideal. After considerable experience in mass radiography with the 14-inch by 17-inch films, Dr. Hallam did not believe that it was a practicable method for the survey of large numbers in a short time. The difference in cost between the microfilm and the 14-inch by 17-inch film was certainly great. One could not reckon this as an insuperable difficulty; but the amount of material necessary to examine, for example, 1,000 to 1,200 men in one day with 14-inch by 17-inch films would be colossal. Those conversant with the radiological examination of a chest would realize the number of cassettes and intensifying screens and the processing facilities that would be necessary, not to mention the extent of the personnel. The space for drying and handling the films in a clearing house and the time consumed in reporting were additional problems. Dr. Hallam found that to report on 30 to 40 large films in an hour was quick working; a speed of 300 or 400 microfilms in an hour could be attained with comparative ease. The microfilm was not so foolproof as the large film, and some pathological changes were undoubtedly missed on it, the main reason being, not that the changes were not detectable, but that monotony and eyestrain became associated with the rapid and orderly succession of images. For example, two cases of basal pulmonary tuberculosis which were discovered on 14-inch by 17-inch films were actually detectable on the microfilms when they were reviewed. The microfilms of these subjects had been passed as clear three or four months before evidence of the disease was found on the large film. Microfluororadiography was a speeded-up pectoral policeman flashing his lamp into every doorway, carrying on further investigation of anything suspicious and finally bringing culprits to a court of inquiry. He was a fallible primary agent of detection, not a censor.

Dr. Hallam went on to say that he had classified mass surveys of lungs into four groups. (i) Military mass surveys, which had been undertaken extensively in many parts of the world; by means of them numerous tuberculous recruits had been precluded from joining the forces. It had been found that five or six in every thousand of such selected persons were suffering from active pulmonary tuberculosis. (ii) Surveys of nurses and students, which were important in any social service. It had been found in one survey in

[1] A series of slides was then shown demonstrating the prolonged inactivity of the round tuberculous nodule—known as the "time-bomb" at the Tuberculosis Bureau. Dr. Hallam said that this might remain inactive for months or years, but finally "exploded" and spattered the surrounding lung with tuberculous nodules.

America that amongst primary school children contacts gave an incidence of tuberculous disease eight times greater than non-contacts of the same age-group; but in the case of adolescents the prevalence of tuberculosis was forty times greater amongst contacts than amongst non-contacts. (iii) Surveys of groups of employees. In a large office in Melbourne several young employees were stricken with pulmonary tuberculosis during two or three years. The chests of about fifty employees were radiologically examined at the request of the firm's medical officer, and it was found that a senior clerk was suffering from bilateral phthisis. Neither he nor anyone else apparently had ever suspected this. (iv) General survey of the population. This would assuredly come in the near future, and already in Melbourne some municipalities were arranging to survey certain age-groups. It had to be remembered, however, that there were innumerable middle-aged and elderly persons suffering from "open" pulmonary tuberculosis, and these lived long to be social menaces for a number of years; they were the greatest danger to the very young and to the adolescent population.

In conclusion, Dr. Hallam said that he had briefly described some methods and had attempted to assess the present status of radiology in relation to pulmonary tuberculosis. The inference was that the practice of medicine was not a slavish allegiance to the dictates of machinery, but was still the art of medicine. For his purpose he would quote Tennyson, that master of the epigram:

> . . . I see in part,
> That all, as in some piece of art,
> Is toil cooperant to an end.

The end was to prevent the escape of tuberculous patients from the drag-net of radiological surveys, to avoid labelling non-tuberculous persons as tuberculous, and to eradicate pulmonary tuberculosis.

Dr. T. E. Lowe showed several spools of microradiographic films by projection from a lantern, and Dr. Hallam commented on them in relation to military work, showing how abnormalities in films were detected. Dr. Lowe supplemented Dr. Hallam's remarks by demonstrating similar features from spools of films taken of civilians at the University of Melbourne in connexion with the experimental work inaugurated by Dr. E. L. Cooper.

Dr. W. S. Newton discussed certain clinical considerations in the early diagnosis of pulmonary tuberculosis. He said that undoubtedly the two outstanding recent advances in the diagnosis of pulmonary tuberculosis were, first, the improvement in radiological technique and methods, and secondly, the correlation of these X-ray findings with the rapidly increasing knowledge gained by the greater use of cultural methods of demonstrating the presence of the tubercle bacillus. The details of these two methods had been ably presented by Dr. Hallam and Dr. Webster. However, in order that their significance should be appreciated from the clinical viewpoint, further reference should be made to them. It was not long ago that radiography of the lungs was a rather unreliable method of investigation; though undoubtedly of help in the diagnosis in some cases, it created confusion in others. This was mainly due to a lack of appreciation of the differences between changes caused by tuberculosis and those caused by other pulmonary diseases. In recent years the improvement of technique and the constant correlation of radiographic appearances with bacteriological and post-mortem findings had demonstrated that certain shadows in certain areas of the lung might now with reasonable accuracy be proclaimed tuberculous.

Mass radiography of the lungs had been carried out in various parts of the world. The two most noted investigations in Australia were those carried out on Broken Hill miners, and the recent work of Dr. H. W. Wunderly, of Adelaide. In the past this work was limited because of the expense of 17-inch by 14-inch films. The introduction of the miniature camera method had removed this obstacle, and as a result Australia was now in the midst of one of the largest investigations of this type ever carried out, namely, the X-ray examination of every recruit for the Australian Imperial Force. It was to be hoped that the investigation would be extended to the Militia and later carried on by the health authorities. Apart from its value in diagnosis, this mass investigation had shown the existence of shadows, almost certainly tuberculous, in persons who had not previously complained of any important symptoms of the disease. Furthermore, serial X-ray examinations over years had shown the varying fate of these lesions. With the knowledge so obtained and with the results of careful clinical study it was now possible to classify the lesions into groups. Kayne Pagel and O'Shaughnessy's classification was, Dr. Newton believed, a most satisfactory one. They classified the lesions into three groups, namely: Group I: those showing no change whatever, and presumably healed; Group II: those showing changes which suggested that the morbid process was progressing or retrogressing; Group III: the latent stationary lesion, giving rise to no symptoms, but containing within it those conditions of bacillus and host that were favourable to recrudescence of the disease, for example, an encapsulated caseous focus. Dr. Newton said that this was the type of case which required to be kept under clinical and radiological review. The radiologist alone could not definitely classify the patients in these groups, but had to be considered as providing only part of the chain of evidence, the physician and the bacteriologist providing equally important parts.

Dr. Newton went on to say that until recently the criteria of healing were that the X-ray findings should be those of Group I. Symptoms were absent. The clinical findings were those of a healed lesion, and either there was no sputum or it was not found to contain tubercle bacilli by the Ziehl-Neelsen method. This conception had received a rude shock since the introduction of cultural methods, not only of sputum, but of the fasting gastric content when suitable sputum was not available. Because of the work carried out by Dr. Webster over the past few years on the culture of the tubercle bacilli, these cultural methods were incorporated with the mass examination of recruits for the Australian Imperial Force in Victoria. Dr. Webster's preliminary reports had recently appeared in *The British Medical Journal* and in The Medical Journal of Australia. His results of 11·1% of positive cultures from subjects whose lesions had been designated radiologically as healed, calcified or inactive, and from whom no symptoms or previous history were obtained, were illuminating, more especially when it was realized that only one cultural examination was carried out on each recruit. Therefore, in order that a lesion should be classified as healed or inactive for practical purposes, not only must it conform with the criteria already laid down, but cultural investigations of the sputum or fasting gastric content of the subject must fail to yield tubercle bacilli on more than one occasion. These results of cultural investigation would also affect the public health viewpoint of the suitability of employment for a subject and the possibility of his proving a source of danger to others. Unfortunately these cultural methods were not simple and had to be left in the hands of capable bacteriologists.

Dr. Newton further said that as a result of these newer methods of diagnosis the physician was confronted with three types of patient, bacteriological investigation of whom was necessary: (i) The patient with suggestive symptoms or signs, or both, who produced suitable sputum. If this did not yield tubercle bacilli by the Ziehl-Neelsen method, it should be inoculated into nutrient media, and if this procedure again failed to produce a result, attempts at culture from the fasting gastric content should be made. (ii) The same type of patient who produced no sputum. In this case an attempt at culture from the gastric content should be made and, if necessary, repeated. (iii) The patient without symptoms, who was discovered by mass radiography or similar means. The procedure in this case would depend on whether or not sputum was obtainable, and in each case should be as already outlined in (i) or (ii).

Dr. Newton then said that so far he had discussed only the help given by these two investigations in the known or suspected case. They could be of little use in the early diagnosis, unless the physician appreciated the signs and symptoms which gave rise to suspicion of pulmonary tuberculosis. Investigation of patients admitted to any sanatorium revealed a far too high percentage in whose cases the diagnosis had been missed for a considerable time, despite the fact that suspicious symptoms were present from the onset. No new clinical methods, as opposed to radiological and cultural investigation, had been postulated; but the improvement in early diagnosis was due to the greater appreciation of those symptoms and signs which should arouse suspicion of the presence of pulmonary tuberculosis. An incontrovertible diagnosis of pulmonary tuberculosis in its early stages was rarely possible, as this presupposed the demonstration of tubercle bacilli in the sputum or fasting gastric content. The process of invasion of the lung was usually slow, and during this time only indefinite symptoms might occur. The degree of toxæmia was slight and might be merely sufficient to cause minor degrees of fatigue and loss of energy and the gradual realization that work and play were becoming rather an effort. It was this very mildness of the symptoms, combined with the fact that mass radiography disclosed many patients with progressive lesions, that had given rise to the well-supported doctrine that the early stage of the disease was symptomless. Dr. Newton did not subscribe to this view. Certainly local symptoms, such as cough and sputum, would not occur until ulceration into a bronchus had taken place. A comparatively small lesion situated near a bronchus

might therefore produce cough and sputum at an early stage, whereas a miliary infection involving many times the amount of lung might not produce those symptoms even in the terminal stage. The amount of toxins circulating in the blood presumably had to reach a certain height before symptoms were produced; but it was not necessary to postulate a high level. In the majority of cases symptoms of toxæmia occurred at a moderately early stage of lung invasion.

The disease usually commenced insidiously, but it might be acute. The acute onset was generally secondary, the primary focus having been present for some time and the acute symptoms being due to the flare-up of an infection which had been previously clinically silent. The early symptoms were mainly toxic in origin, depending on the liberation of toxins from the bacilli and the destroyed lung tissue. The stage of their appearance depended not only on the extent and situation of the lesion, but also on the resistance of the patient. These toxic symptoms were indefinite and consisted of malaise, undue fatigue, the feeling that the day was too long, loss of appetite, nervous upsets and dyspnœa; or there might be a story of frequent colds indicating a spread of the intrapulmonary lesion and the release of fresh doses of toxins. Persistence and progression of such constitutional symptoms should cause the physician to suspect pulmonary tuberculosis, more especially if persistent pyrexia or tachycardia coexisted. In many cases the symptoms did not direct attention to the lungs, and the signs might be absent or so indefinite that the lungs were not suspect. Difficulty arose, first, because pulmonary tuberculosis was not thought of, and later, because it was not sought for sufficiently vigorously. A further difficulty was that these toxic symptoms were common not only to pulmonary tuberculosis, but to many other conditions, such as focal infections, or they might be merely the result of stress and strain, overwork or psychological disturbances. The physician therefore had to distinguish between symptoms due to disease and those due to the patient's habits and mode of life. In this regard a careful history was helpful, particularly if it included thorough inquiry as to the mode of life of the patient over the preceding few months, and it should comprehend careful interrogation as to increase in work and the occurrence of psychological upsets. Recent advances in investigational methods had in no way detracted from the value of history taking; in fact, when intelligently and carefully obtained, the history formed the most valuable approach to the diagnosis of pulmonary tuberculosis in its early stages. It should include inquiry into the family history. If certain members of a family were susceptible to tuberculosis, it was reasonable to assume that all members were equally susceptible. The history of contact with an "open" case might provide the clue to the diagnosis. The past history was of great value. A history of surgical tuberculosis was evidence that the patient had suffered from chronic disseminated tuberculosis, a protracted stage of tuberculosis characterized by healing in one part and the development of an active lesion in another. Pulmonary lesions were quite common in this phase, though they might be latent and not become manifest until much later. *Erythema nodosum* was often said to be due to tuberculosis, but it was probably an allergic reaction to bacterial toxin. *Mycobacterium tuberculosis* might be the origin of the causative toxin, as it appeared to be in Norway and Sweden; but so might many other organisms. The presence of an ischio-rectal abscess should be regarded as presumptive evidence that an associated pulmonary lesion was tuberculous, though an ischio-rectal abscess need not be tuberculous in origin. A history of pleurisy was important; but care had to be taken in the assessment of the value of a history of dry pleurisy. This might occur in association with many other lung conditions, and the diagnosis was often erroneously made when there was any complaint of pain in the chest. Pleurisy with effusion, if the diagnosis had been proved, had a very different significance. In the majority of cases it was due to tuberculosis, despite the fact that the fluid might be sterile and the X-ray findings negative. In many cases pleurisy was caused by subpleural tubercles, the presence of which was hidden by the effusion in the earlier X-ray films, and if these healed coincidently with absorption of the fluid they might not be demonstrable in the later films. Dr. Newton looked upon a past history of effusion as an indication for constant clinical and radiological observation for at least two years.

Dr. Newton went on to say that a recent history of influenza, especially if the patient had never completely regained his health since the attack, or if a persistent cough had developed, should be considered suspicious. On the other hand, every patient with "influenza" whose symptoms did not subside in a few days or who developed an associated bronchitis or bronchopneumonia which persisted unduly, should have the sputum examined. The routine X-ray examination of all patients convalescent from influenza would bring many subjects with tuberculosis under treatment at an early stage. Undue tiredness and loss of energy and interest in work might be due to a variety of causes, one of which was neurasthenia. This condition should not be diagnosed until all the possible organic causes, one of the commonest of which was tuberculosis, had been eliminated. A cough or bronchitis which persisted should always arouse suspicion. It might be due to pharyngitis caused by excessive smoking—the so-called cigarette cough—or it might be due to an upper respiratory tract infection. The latter condition, especially if an associated bronchitis developed, might closely simulate pulmonary tuberculosis, and this could not be excluded unless X-ray and bacteriological investigation produced negative results. The sputum should be tested in all cases of chronic bronchitis. In 1922 Dr. Newton had carried out an investigation of the sputum in all cases labelled as "chronic bronchitis" in an out-patient clinic, with the result that 17% of specimens were found to contain tubercle bacilli. With the present-day cultural methods Dr. Newton thought that this percentage would have been greatly increased.

Dr. Newton further said that the occurrence of hæmoptysis should always indicate complete investigation. The more comprehensive knowledge of chest pathology gained in recent years had shown many causes of this symptom; but tuberculosis probably still held pride of place. The old story that its source was the pharynx or larynx was still prevalent. Unless the bleeding point was seen, the lung must be held suspect and appropriate investigations instituted. Persistent vague digestive symptoms, especially if they were associated with loss of weight and if examination revealed no gastro-intestinal lesion, should arouse suspicion of pulmonary tuberculosis. How often had an expert radiologist discovered the cause of the symptoms to be above the diaphragm when conducting an opaque meal examination! The history of patients with advanced disease of the lung showed that many of them were treated for long periods for indigestion or neurasthenia before the true diagnosis was made. Amenorrhœa was a common symptom of pulmonary tuberculosis, and it might occur at an early or late stage. If it was not due to physiological causes, this symptom demanded a pulmonary X-ray examination. Persistent pyrexia and tachycardia without obvious cause were indications for thorough investigation.

Dr. Newton went on to say that it was not within the scope of his remarks to discuss physical findings in detail. He hoped that by frequent mention of radiological and bacteriological investigations he had not given the impression that physical signs were of minor importance. Unfortunately there existed an idea amongst some physicians who placed almost their entire trust in "machine tools", such as the X-ray examination and the electrocardiogram, that these physical findings were of little importance; equally unfortunately, there still existed some physicians who subscribed to the doctrine: "No physical signs, no tuberculosis." No physician could afford to neglect physical findings in diseases of the chest, and in many cases they were of paramount importance. Physical signs might be absent or indefinite in pulmonary tuberculosis, not only in early cases, but also in moderately advanced cases. It often happened that one saw patients who presented the symptoms of cough, pyrexia and wasting due to an acute tuberculous lesion, with signs so vague that, on physical examination, the diagnosis was extremely difficult. As Morriston Davies had stated, no sign was pathognomonic, nor was any individual sign of value. It was the sum total of all the physical findings, and the interpretation of what they represented in terms of pathology, together with the history and symptoms, that constituted the grounds for suspicion of a tuberculous ætiology. The value of the Mantoux tuberculin test in diagnosis was still a matter of considerable difference of opinion. The Price protein derivative should be used, and the result could not be regarded as negative unless the test was carried out with a dilution of 1 in 100. It was no doubt of great value in diagnosis of the disease in young children; but in adult practice its only virtue was in the comparatively rare negative result. Dr. Newton considered the blood sedimentation rate to be valueless in diagnosis, but it might be of some help in the management of the patient.

Dr. Newton then stated his conclusions in the following form: (i) that the early diagnosis of pulmonary tuberculosis depended on team work between the physician, radiologist and bacteriologist; (ii) that modern radiological and bacteriological methods were a great advance in the establishment of a diagnosis; (iii) that if the greatest advantage was to be obtained from these advances, the physician had to be on watch for those signs and symptoms which should arouse suspicion and which demanded investigation; (iv) that if proper appreciation was given to these symptoms, much valuable time and many valuable lives would be saved.

Dr. H. M. James discussed the tuberculosis officer in relation to diagnosis. He said that he felt honoured at having been asked to take a part in the symposium, which was one of great importance in view of recent developments in the diagnosis of tuberculosis. He first of all explained the work of a tuberculosis bureau. The Central Tuberculosis Bureau had been established for the benefit of those unable to afford private or intermediate fees, and patients were seen only on the recommendation of a medical man. On the clinical side, for the tuberculosis officer there were three fairly well-defined duties: (i) the provision of a consulting service in doubtful cases of pulmonary disease; (ii) the supervision, both before and after a stay in a public sanatorium, of the patient with an established tuberculous lesion in the lungs; (iii) the examination and general supervision of the contacts of these patients. From the consultant point of view anything from 40 to 80 patients were referred each week for an opinion. Naturally not all—in fact, very few—were suffering from active pulmonary tuberculosis. This, Dr. James thought, was a matter for congratulation rather than otherwise, in that it served to indicate that many medical practitioners were alive to the necessity of further investigation of their "doubtful" cases.

Dr. James went on to indicate briefly the bureau's ideas of what should constitute a comprehensive pulmonary investigation. With regard to the history, he was certain that many patients referred to the bureau had no actual organic disease. Their disability was the result of antecedents, environment, habits, occupations, personal tendencies and even domestic difficulties. It was no use whatever, examining a patient's lungs and considering one's duty done. The patient had to be seen as a whole, and not the patient alone, but everything possible pertaining to him. Unless they were prepared to dig, and to dig deeply, into the personal and family history, Dr. James thought they were foredoomed to failure. And just as carefully as they investigated environment, they should search for possible contact with tuberculosis. It was astonishing how many unsuspected cases of phthisis could be found if the investigator was sufficiently persistent.

The basis of all tuberculosis work was prevention, and for that reason the first thoughts of a tuberculosis officer on meeting a patient suffering from tuberculosis should be: where had he come from? There had to be a source of infection somewhere; tuberculosis was an infectious disease, and there must have been some point at which the patient became infected. At times, as they knew, the condition might be one of resurrection of a childhood infection, and the original infecting patient might have died many years previously. Too often, however, there was still a potent source of infection somewhere about and often unsuspected. Dr. James was referring to the carrier type (and the presence of a carrier type in tuberculosis had to be admitted); this was often the elderly relative or friend in the home, who enjoyed perfect health, but admitted having a cough in the winter time. Time after time such people had been found, expectorating sputum loaded with tubercle bacilli, and radiologically showing old, chronic, thick-walled cavities. Nothing much happened to them; but they constituted an ever-present danger. Certainly the whole family should be investigated; but the aged menace sitting comfortably in the arm-chair by the fire should not be forgotten. Dr. James pointed out that in diagnosing tuberculosis in one member of a family the tuberculosis officer more or less accepted the responsibility for investigating all the others.

With regard to personal history, Dr. James said that pleurisy with effusion in the adolescent was accepted without reserve as of tuberculous origin. True, a number of young people recovered completely and showed no further signs; but on the other hand, an enormous number did develop clinical pulmonary tuberculosis within two or three years. Patients suffering from influenza, *fistula in ano*, supposed chronic duodenal ulcer, neurasthenia including irritability of temper, and *diabetes mellitus*, should all be watched carefully, because of the frequent association of these conditions with phthisis. Dr. James thought it hardly necessary to apologize for mentioning hæmoptysis, although the differential diagnosis of this symptom had been familiar to everybody for years. Certain well-known conditions were associated with hæmoptysis—pulmonary tuberculosis, mitral stenosis, pulmonary hydatid cyst, lung abscess and neoplasm; but in the adolescent, and particularly when the hæmoptysis appeared suddenly in a patient in otherwise comparatively good health, there was little doubt that the condition was tuberculosis. For years Dr. James had been hopefully, but unsuccessfully, looking for varicosities of the throat and œsophagus.

Dr. James next referred to physical examination. He said that two questions were often asked: Was it reliable? Was it worth while, when there was such a valuable source of information readily accessible as the X-ray examination? Dr. James thought it was. The careful physician was competent to diagnose phthisis in certain cases before the appearance of radiological signs. If there was a sufficiently suggestive history, family and personal, backed by a positive response to the Mantoux test, what were the earliest physical signs to be expected? A little lag, a slowness of expansion on one side, coupled with an increase in muscle tension over the shoulder girdle on one side, should provide early and valuable clues. These might all be present before radiological signs were apparent, and in the presence of the right history justified a diagnosis of pulmonary tuberculosis to the physician himself, even if he did not immediately convey such an impression to the patient. The evening rise of temperature was well known; but of just as great importance was the daily variation of temperature, which should not be much more than 1° F. Dr. James thought that the regular premenstrual rise of temperature in young women could be ignored; but if this was exaggerated and prolonged it should be regarded with some degree of suspicion. Endocrine disturbances were also not unusual in young women in early phthisis—excessive mammary development, or an enlarged thyreoid gland without evidence of thyreotoxicosis, in addition to the usual amenorrhœa.

Dr. James went on to say that once it was suspected that a patient was suffering from tuberculosis, the next moves must immediately be examination of the sputum and radiography, both of which had been comprehensively dealt with by Dr. Webster and Dr. Hallam respectively. With regard to the sputum, however, he suggested that concurrently with the examination for *Mycobacterium tuberculosis* a search should be made for other organisms, particularly the yeasts. Moniliasis was by no means uncommon, and might cause radiological appearances suggestive of tuberculosis. At the bureau they had seen one case of torulosis in which a vague generalized mottling was noted in the lung fields. On the radiological side, new problems had been greatly emphasized by the recent development of mass radiography. Many unsuspected lesions had been found, and in a large number of instances the patient would not admit, even on searching cross-examination, to anything at all in the way of past history or present symptoms. From the radiological appearance of the lesions it was impossible to be certain that no activity existed. Clear-cut calcified lesions had, in many cases, been accompanied by the production of sputum containing tubercle bacilli even in the direct smear, and, as Dr. Webster had said, in many more cases when sputum or fasting gastric content had been subjected to modern cultural methods. They might ask whether the presence of a positive culture necessarily denoted tuberculous activity. So far at the bureau it had been possible to watch some of the patients over a period of nearly twelve months, and they were agreed that tuberculous activity and the presence of *Mycobacterium tuberculosis* in the sputum or gastric content went hand in hand. The activity might be of low-grade type, but it was undoubtedly there, and in most cases progressive. With regard to the radiological appearances, Dr. James said that he would not accept any lesion in the adolescent as healed or even quiescent, no matter how calcified it looked, without a long period of observation, and that he would similarly not accept as quiescent without observation any lesion whose complete level was below the area of the clavicle. He knew of no easy way to determine the activity or otherwise of a tuberculous lesion. It was only by careful plodding and painstaking observation over a period that medical men could even for themselves alone approach a definite conclusion.

With regard to special investigations, the first to be mentioned was the tuberculin test—the Mantoux and the Vollmer or patch test. At the bureau they had been using Anderson's "one-shot" dose for the Mantoux test, in which the actual dosage was 0·00125 milligramme of "P.P.D." ("purified protein derivative"). Dr. James did not think a smaller dosage was reliable. He also thought that, in order to be quite certain, in the absence of a reaction the test should be repeated in from seven to ten days, so that errors in technique or defective tuberculin might be excluded. At the bureau it was considered that an absence of reaction to a test carried out in this way excluded the possibility of an active tuberculous infection. Two interesting instances of this had occurred recently. The first patient, a boy, aged twelve years, the son of a returned soldier suffering from proved pulmonary tuberculosis, was reported as having intermittent febrile attacks, and an X-ray examination revealed a fan-shaped area of mottling in the upper lobe of the left lung. The diagnosis appeared to be obvious until the child was thoroughly investigated in a sanatorium, where the Mantoux test repeatedly failed to produce a reaction. The diagnosis was apical pneumonia. The second patient, a boy, aged sixteen years, whose mother had had a condition thought to be pulmonary tuberculosis for many years.

although it was not proved, had repeated brisk hæmoptyses. An X-ray examination revealed extensive mottling throughout almost the whole of the lower lobe of the right lung. Again the diagnosis appeared to justify the patient's admission to a sanatorium; but the tuberculosis officer who was looking after him was of a persevering type of mind, and continued to carry out tuberculin tests, which repeatedly failed to produce reactions. The diagnosis was lung abscess.

Dr. James said that the Vollmer or patch test was simply performed, and for that reason particularly suitable for children. The patch was easy to make and easy to apply, and if necessary it could be placed in the interscapular area, where it could not be reached by the child. The result was best read at seventy-two hours. The details of the test had been published in a recent issue of THE MEDICAL JOURNAL OF AUSTRALIA. Dr. James suggested that it was not always wise to tell an anxious mother the nature of a tuberculin test. In spite of all that she might be told, she might remain convinced that her child was actively tuberculous if she saw a positive reaction.

Speaking of the blood sedimentation rate, Dr. James said that its estimation was useful and interesting, but of doubtful value in diagnosis. It had to be remembered that pulmonary tuberculosis was a disease of ups and downs, of exacerbations and remissions, rather than one prolonged course of either recovery or decline. The blood sedimentation rate during remissions might be quite normal, and in fact tuberculosis of low-grade activity was often associated with a normal erythrocyte sedimentation rate. The performance of a Wassermann test was essential; a gumma might be indistinguishable radiologically from a neoplasm. Dr. James was also convinced that in certain cases of syphilis the X-ray film of the lung presented some rather characteristic features—a peculiar spider-web appearance, more pronounced near the hila and lessening towards the peripheral part of the lung. In addition, a Casoni and a hydatid complement fixation test were suggested; Dr. James pointed out that hydatid cysts might be surprisingly atypical in appearance at times. Certain of the blood diseases, purpura and leuchæmia, were at times accompanied by a congested appearance of the lungs and in that way simulated miliary tuberculosis. Dr. James had himself admitted to a sanatorium a patient suffering from myeloid leuchæmia instead of miliary tuberculosis. Thus in a doubtful case a differential blood count was suggested. Occupation also had to be considered. If the patient had been a miner, an asbestos worker or an electrical welder, any of these callings might be responsible for diffuse mottling in the lungs. Lung abscess was usually easily eliminated from the diagnosis by the clinical history. Congenital cystic lung had to be diagnosed radiologically with the aid of tomography and possibly opaque oil.

Dr. James went on to say that it was necessary to eliminate an extrathoracic cause for any pulmonary symptoms. A fair proportion of patients sent to the bureau were suffering from chronic sinusitis with associated bronchitis. A noticeable feature of many such cases was that an anxiety state was frequently present as well—much more so than in the case of actual pulmonary tuberculosis. This led to an accentuation of symptoms, which might cause the patient to believe that he was suffering from tuberculosis. In any case, a thorough investigation of the upper respiratory tract should be included in the examination. Bronchiectasis might cause no radiological signs, but clinical signs were usually present. Adenocarcinoma might cause profuse bleeding without any obvious signs. Bronchoscopic examination, with or without opaque oil, was the accepted next line of attack. The diagnosis in many such cases, however, was not easy, and required patience on the part of the physician and tolerance on the part of the patient. Negative findings did not necessarily exclude an eventual diagnosis of tuberculosis. In the meantime continued observation should be persisted with; records should be kept of the temperature, weight, response to exercise, and so on, and serial X-ray examinations should be carried out if possible every month or two. If the patient was tuberculous, the observation period would do more good than harm. Dr. James then uttered a word of warning. He said that once a diagnosis had been established, for example, of lung abscess, unresolved pneumonia, cystic lung, silicosis or any similar condition, continued vigilance was still necessary. Tuberculosis might be superadded to almost any of these conditions at any stage. At the bureau they had recently been surprised by the appearance of tubercle bacilli in the sputum of two patients suffering from unresolved pneumonia; and at the Royal Melbourne Hospital there was a patient suffering from congenital cysts, and tuberculous cavities were present at the same time.

Dr. James said that as a tuberculosis officer he viewed with great relief, as an undoubted step upwards, the advent of mass radiography and intensive sputum examination. Up to one month before the meeting 431 men had been referred to the bureau from the army alone, radiological examination having revealed tuberculous lesions. Roughly, about one-third of these men had been proved to have lesions of some degree of activity; yet all the men had been passed as fit on ordinary clinical examination. Dr. Webster had given the comparative figures from the testing of sputum. Dr. James was prepared to make the statement that, in his opinion, not one-quarter of these men would have stood up to the stress and strain of active service. During eight years which he had spent at the Victoria Repatriation Sanatorium after the first world war, over 1,300 ex-soldiers had passed through his hands, all suffering from pulmonary tuberculosis, and the great majority accepted as eligible for pensions or with lesions due to war service. Many of these men had seen very little service; some had not heard a shot fired; yet all were pensioned. Dr. James thought that such a state of affairs should not obtain after the present war; mass microradiography should save many hundreds of thousands of pounds.

Dr. James went on to say that at the bureau they had carried out what might be regarded as routine radiological examination of contacts of their tuberculous patients. In a series of about 1,000 tuberculin tests among contacts, about 80% were found to produce positive results. All persons who reacted to the test had to be radiologically examined, and of the 20% or so who failed to react, many were radiologically examined for one reason or another. In a series of approximately 1,700 contacts examined a few years earlier, they had found 11% giving radiological evidence of lesions. That was far and away above the figures found in radiological surveys of the ordinary civilian population. Dr. James considered that the culture of sputum and fasting gastric contents would without doubt save an enormous amount of work. He remembered one occasion on which tubercle bacilli were found at the forty-third examination of sputum. The lesson learned from earlier experiments in mass radiography was the desirability of extending this form of investigation to all classes of the community. If 431 men in the army, who were thought to be physically fit, were found radiologically to have tuberculous lesions, then there would be at least the same number of young women walking about with similar undetected lesions. Wunderly's work at Adelaide left no doubt that this was so. At the tuberculosis bureau they had been disappointed that mass radiography of the citizen forces and Militia had not been carried out. On several occasions already soldiers attempting to transfer from the Militia to the Australian Imperial Force had been found to be suffering from active pulmonary tuberculosis, and it was obvious that many more would still be present and undetected. They must serve as a source of infection to their camp mates and fellow soldiers, and Dr. James felt that an investigation was urgently needed in that sphere.

Dr. James then said that sanatorium superintendents continually asked that patients be sent to them in a reasonably early stage of the disease. Undoubtedly too many with advanced and chronic lesions were in the sanatoria; the blame might be apportioned between medical men and the people themselves. At the present time there were ample facilities for the complete investigation of persons with pulmonary lesions, even of those who could not afford private consultation, and as medical men they should see to it that advantage was taken of those facilities. If they did not do so, they were "falling down on the job". Unfortunately, the patients themselves did not report for examination in the early stages. They did not feel ill and saw no necessity for undergoing medical examination. Until mass radiography of all groups of the community was established, that state of affairs would persist, and the sanatoria would be choked with patients who had advanced and chronic lesions, for whom the chance of alleviation was only slight and the prospects of cure were nil. Under a properly organized system of mass radiography it would be possible gradually to collect all patients in the stage of minimal lesions. Dr. James liked the story of the medical officer of health in one of the South American States, who was given a totally inadequate amount of money for his anti-tuberculosis scheme. Not being able to build sanatoria *et cetera*, he established in various parts of the country X-ray plants and made the service free to the people. He took the attitude that a person with a known lesion was less danger to the community than someone with an unrecognized lesion, and that by acting as he did he had put down the first plank in his platform of prevention. In conclusion, Dr. James said that if it were possible to have mass radiography of the whole population, supported by intensive sputum search in doubtful cases and backed by an adequate pension for the tuberculous family bread-winner, then all but the minor problems of tuberculosis would be solved.

DR. K. STUART CROSS said that he thought certain qualifications should be kept in mind in regard to the elliptical phrase "X-ray findings negative", as used in the tables presented by Dr. Reginald Webster, whose work the speaker had followed with the greatest interest and admiration. The term referred to should be considered as usually meaning only that in a single film of the chest no tuberculous changes were seen or recognized. Quite apart from the technical quality of an X-ray film, its interpretation was a highly individual matter. Quite recently three experienced radiologists sat together in critical examination of a film of a patient's lungs taken several months previously. One of them considered that certain relatively faint markings in the subapical portion of the upper lobe of the left lung were not part of the linear branching bronchovascular pattern and probably indicated early tuberculous parenchymatous infiltration, and in fact had reported to that effect, recommending reexamination after a reasonable interval of time. The other two thought the appearance was probably of no clinical significance and not outside the limits of the healthy adult lung. A second film of the same patient, taken several months later, was then exhibited. It showed gross and widely spread tuberculous changes in and around the area indicated.

Even skiagrams made on films of equally good quality, taken of the same patient, but with different radiographic techniques, could lead to different conclusions. Early lesions lying in front of or behind ribs, or perhaps both, or in the most unfavourable case with the superaddition of the clavicle, could easily escape detection, particularly in films taken without the Potter-Bucky diaphragm. In that case either the scattered radiation reduced contrast or, to save the latter, soft, relatively non-penetrating rays were used, so that parenchymatous changes obscured by bone were not registered. Then again, many films were not of a sufficiently high technical quality. That applied particularly to the miniature films used in the fluorographic examination of army recruits *et cetera*. In that case probably less than 50% were of really good quality, owing to errors in exposure, poor processing, or both. One had to assume that a number of early lesions escaped detection. It was not logical, therefore, to deduce that certain early stages of tuberculous involvement of the lungs were incapable of radiological demonstration merely because in certain individual cases they had not been detected.

DR. KENNETH SMITH, after expressing his appreciation of what had gone before, commented on the use by Dr. James of the word "pension". Dr. Smith deprecated the use of the word in that connexion, especially when applied to civilian requirements. He recommended that the payment should be called a treatment allowance, on account of the idea of permanency which the word pension carried by usage. The term "treatment allowance" conveyed the sense of the temporary nature of the payment and of the obligation imposed upon the patient to undergo treatment. Finally, Dr. Smith urged support for those who were pressing for thorough investigation of tuberculosis amongst the Militia establishments.

The Acting President emphasized the peril from the exposure of babies and children to contact with older sufferers from tuberculosis. The serious consequences to the young members of the community were evident unfortunately only too often in every medical practice, and it was urgently necessary that effective methods of prevention should be organized. After he had expressed the appreciation of the audience to the opening speakers and commented on the high quality of their contributions, he invited them to add any comments they wished, but as each of them indicated that he had nothing further to say, the meeting was concluded.

A MEETING of the South Australian Branch of the British Medical Association was held on July 31, 1941, at Adelaide, DR. R. J. VERCO, the President, in the chair. The meeting took the form of a series of clinical demonstrations.

Stones in the Common Duct Dissolved by Ether.

DR. A. C. MCEACHERN showed a female patient, aged forty-two years, who had been admitted to hospital on April 27, 1941, with a history of epigastric pain and jaundice of three weeks' duration. During this time she had had three typical attacks of biliary colic. There had been no previous attacks or dyspepsia.

Examination revealed a moderate degree of obstructive jaundice, associated with enlargement of the liver below the level of the umbilicus and enlargement of the spleen. X-ray examination revealed that the right side of the diaphragm was normal. The gall-bladder was not visualized after injection of dye. The hæmoglobin value was 14·82 grammes *per centum* and the blood urea level 14·0 milligrammes *per centum*. The Casoni test for hydatid produced no reaction.

Preoperative treatment comprised the administration of vitamins *A*, *C*, *D* and *K*, ox bile, glucose and an increased amount of fluids. At operation, under ether anæsthesia endotracheally induced, the liver showed the changes of biliary cirrhosis. The gall-badder and all the accessible bile ducts were found packed with faceted stones. The gall-bladder was opened, evacuated and drained, and through a tedious exploration of the common duct as many stones as could be coaxed down from the hepatic ducts were removed, and the lower end of the common duct was shown to be patent. The common duct was drained.

At the twelfth day the stools were still clay-coloured, suggesting an obstruction of the common duct. The gall-bladder drainage tube was clipped and lipiodol was introduced into the common duct. This allowed the gall-bladder and the common and hepatic ducts to be visualized, and revealed a block in the supraduodenal portion of the common duct. A duodenal tube *in situ* aided the interpretation of the X-ray appearances.

Five cubic centimetres of ether were introduced through the common duct tube every day for three days and the tubes were clipped off for an hour. This caused no pain or discomfort, the colour of the stools rapidly improved, and the patient remained well.

Arterio-Venous Aneurysm and Gas Gangrene.

Dr. McEachern also showed a male patient, aged sixteen years, who had been admitted to hospital on April 14, 1941, suffering from a 0·22 calibre bullet wound of the right thigh, sustained on the previous day. There had been very little external bleeding. The entrance wound was over the femoral vessels in the middle third of the thigh. A diffuse swelling associated with a thrill suggested the likelihood of an arterio-venous communication; this was confirmed by the presence of a peculiar thrill over the femoral vein at the groin and by blood pressure readings of 145 millimetres of mercury systolic and 36 millimetres diastolic.

With a view to possible arterial and venous repair the continuous administration of saline solution containing 5,000 units of heparin per litre was begun. Antitetanic serum and 4,000 units of gas gangrene antiserum were given.

At operation on April 15 the vessels were exposed in Hunter's canal and controlled with fine tourniquets above and below. A direct communication existed between the femoral artery and vein, but the extent of the damage and the presence of troublesome bleeding from collaterals precluded the planned end-to-end anastomosis. Artery and vein were ligated above and below the injury and the damaged segments were excised.

On April 16 the patient's temperature rose to 102·6° F. and the pulse rate to 104 per minute. The skin became discoloured, there was a profuse, musty-smelling discharge from the wound, and a little crepitus was felt. The next day X-ray examination revealed gas in the tissues, and exploration revealed infective gangrene confined to the sartorius muscle, especially in the three inches distal to the bullet track. The whole of the sheath from origin to insertion was infiltrated with dark blood. The entire muscle was excised and 30 grammes of sulphanilamide were placed in the wound, which was loosely sutured. Sulphapyridine (26 grammes) was administered over a period of seven days and a course of deep X-ray therapy was given. Rapid and complete recovery occurred. Bacteriological investigation both before and after operation revealed no organisms. Examination of the excised muscle revealed acute purulent myositis with extensive degeneration of the contractile substance. Dr. McEachern said that Professor Platt was again examining the histological material for organisms, and his present comment was that the negative findings did not preclude a diagnosis of gas gangrene.

Treatment of Varicose Veins by High Ligation, Section and Injection.

DR. OWEN M. MOULDEN showed a series of five patients suffering from varicosity of the *saphena magna* who had been treated by the method of high ligation, section and injection, which he had been using during the last six months. Dr. Moulden said that the patients were unselected, and he exhibited them to demonstrate the uniformly satisfactory result of the use of this method. He went on to describe details of the technique that he used.

Under local anæsthesia by the infiltration of about twenty cubic centimetres of a 2% solution of "Novocain", with the addition of five minims of a 1 in 1,000 solution of adrenaline chloride, an incision, between three and four inches in length, was made in the groin, immediately over the bulb

of the *saphena magna*. Dr. Moulden stressed the importance of the accurate localization of the incision, failing which the dissection of the main trunk and its tributaries was difficult. The dissection of these vessels having been completed, each in turn was ligated in two places and cut between the ligatures. An injection of sclerosing fluid was then made downwards into the main trunk of the *saphena magna* below the site of section, and a ligature was then placed below the site of puncture to prevent leakage outside the vein. Immediately the injection had been made Dr. Moulden carefully massaged the sclerosing fluid downwards along the course of the vain to its origin in the foot. He used "Ethamolin" as a sclerosing agent. He had used between seven and fifteen cubic centimetres of this, according to the capacity of the saphenous tree. It was usual to obtain a complete thrombosis, at least to the level of the ankle joint, within a few minutes. The patients were allowed to go home immediately after operation and were instructed not to keep the limb at rest.

Local and general reaction following operation varied considerably. Some patients had a sufficiently severe general reaction to necessitate as much as three days in bed. Others had little general reaction. The local reaction usually consisted of extensive perivenous redness and tenderness with staining along the whole course of the venous tree. The period of disability following the operation varied. It was usual for a healthy adult patient to be able to resume his occupation in from twelve to fifteen days. The immediate subjective and objective results were dramatic. Dr. Moulden said that he showed his patients only to demonstrate the technique that he was using and the immediate results. He hoped at a later date to be able to describe the end results of a fairly large series.

MEDICO-POLITICAL.

A meeting of the Victorian Branch of the British Medical Association was held on August 6, 1941, at the Medical Society Hall, East Melbourne, Dr. H. Boyd Graham, D.S.O., M.C., the Acting President, in the chair. The meeting was called for the purpose of altering rule 7a.

Alteration of Rule.

It was decided that rule 7a should read as follows:

> Non-payment of Subscriptions: If the subscription of a Member for any year shall not have been paid on or before the 31st day of December in that year, he shall *ipso facto* (unless any ethical enquiry as to his conduct is pending) without prejudice to his liability to the Association, cease to be a member as from that day. Provided that upon payment before the 31st day of March in the succeeding year of all subscriptions due from him he shall, if eligible, be restored to membership without re-election.

Correspondence.

HARARA AMONG AUSTRALIAN SOLDIERS.

Sir: The following brief note on the occurrence of *harara* among Australian troops in Palestine may prove of interest. *Harara* is the Arabic name for a skin eruption common in Palestine during the months of May, June and July, especially among those newly arrived in the country.

Eight days after a small body of troops, new to the area, moved at the latter end of May into a wood in northern Palestine heavily infested with sandflies (*Phlebotomus* species), the first two cases presented themselves, to be followed daily by others. Their faces and the exposed portions of their extremities were covered by intensely hard wheals up to one centimetre in diameter. In one or two days the wheals subsided, to be replaced in some cases by blisters containing a clear watery fluid under considerable pressure. In these the contents later changed, owing to secondary infection, to a thick yellow pus, and the area involved increased in size until it was in some cases as much as four centimetres in diameter. Gradually the exudation decreased and the areas became comparatively dry and encrusted, though still oozing at one or two points. As long as four weeks elapsed after moving from the area before the lesion completely healed. The degree of severity varied enormously. In some cases the regional lymph glands became very swollen and tender.

The connexion between the skin eruption at the urticarial stage and phlebotomus bites is by no means obvious, and is even less so at the secondarily infected stage, so that the first cases seen may prove extremely puzzling to the uninitiated.

Dr. O. Theodor has shown clearly (*Transactions of the Royal Society of Tropical Medicine and Hygiene*, Volume XXIX, November, 1935, page 278) that the wheals and blisters are successive reactions appearing at the site of phlebotomus bites during the process of sensitization, and has defined *harara* as "the reaction to phlebotomus bites at the height of sensitization". Those interested will find in the paper quoted an excellent account of the condition and a very complete bibliography.

Yours, etc.,

Ronald K. Macpherson,
Staff Sergeant.

Australian Field Hygiene Section,
Second Australian Imperial Force,
Abroad.
July 14, 1941.

THE PSYCHIATRIC EXAMINATION OF RECRUITS.

Sir: The current comment on the psychiatric examination of recruits published in the journal of July 26, 1941, expresses much the same views on this subject which I already had in mind to put in the form of a letter to you.

Sufficient of World War II is over to enable us to form an opinion as to whether phychiatric examination of recruits from the outset would have been profitable and whether it is desirable even at this stage to seek trained assistance in assessing individual suitability for modern warfare. There has been some confusion of ideas between the conceptions of phychological ability and psychiatric suitability. Certain branches of the Navy, the Royal Australian Air Force, and, more recently, the Army, I understand, make use of simple tests for practical ability, reaction time *et cetera* in the selection of personnel for entry or promotion. In Sydney facilities are available to a limited degree for the skilled psychiatric assessment of men seeking entry to the Navy; but there has been a lack of inspiration and direction from the services in respect to the provision for and application of such an inquiry. In most cases, providing a recruit fulfils satisfactorily the physical criteria set down for entry, it is often difficult to reject him on grounds of psychiatric unsuitability, even if one is capable of estimating this correctly. Recruiting, for the Army especially, is often of necessity undertaken by medical officers of all shades of experience and all degrees of familiarity with the red lights of psychiatry, and we see ophthalmologists, laryngologists and obstetricians standing alone in the medical doorway to the fighting services, and expected to judge with equal accuracy of the psychiatric constitution of the youths passed rapidly before them.

As your contributor states, it is not practicable to provide for a full psychiatric examination of each recruit, ideal though this would be; but if there is suspicion of emotional instability, the candidate should be submitted to at least half an hour's investigation by a competent authority. If half an hour's investigation is ultimately going to save the individual from permanent mental invalidism, and the community from the provision of another permanent pension, the trouble would be well worth while. Some instruction of each recruiting officer either by pamphlet or, better, by the corresponding psychiatrist, would reduce the volume of men so referred.

Having examined over 3,000 recruits for the present war, I agree that no reliance can be placed upon the statement of a keen young man in respect to his previous health, even when given in writing, and under threat of a penalty such as forfeiture of post-war benefits. He is more likely than ever to conceal the fact of a nervous breakdown, of traumatic neurasthenia, or of an unfortunate family history. The recruit's regular medical attendant is not necessarily able to speak with authority upon the lad's mental make-up, but may be able to give valuable information.

Expressions such as "this is entirely a young man's war" are both stupid and dangerous. No arbitrary age can be selected to divide the young from the old, neither in the services nor in the civilians of London, for example. The question is essentially an individual one. Ultimate conduct under strain must always remain to some degree unpredictable. None the less, an organization which includes opportunity for a skilled psychiatric opinion must produce better results. Recruits about whom some doubts are sustained can always be accepted for home or base duties.

Yours, etc.,

H.M.A.S. *Westralia*,
July 31, 1941.

Kempson Maddox.

ON THE NATURE OF ECLAMPSIA.

SIR: Captain J. M. Kelly alleges (October 4, 1941) that I have confused cause and effect in dealing with the nature of eclampsia.

I would like to dispute this because in the section of my paper dealing with the treatment of the declared disease I pointed out that the clinical picture of eclampsia is readily demonstrated in rabbits. If small doses of sarcolactic acid are injected every five minutes, the rabbit will become comatose and have convulsions. It is difficult, therefore, for me to see how I have confused cause and effect.

He further states that I have brought forward no evidence in favour of my theory of infection as a cause of eclampsia. I did bring forward evidence that two rabbits of Goldblatt, of London, and a series of toxæmic rabbits of Corkill, of Melbourne, showed an absence of glycogen in the liver, so that either a bacterial infection or a toxæmia apparently can cause this failure to produce glycogen, and consequently the sarcolactic acid in the body cannot be built up to glycogen in the liver. My theory will depend upon the results of alkaline treatment in practice.

I agree with Captain Kelly in his estimate of the "eliminative" treatment, and I would suggest that heavy doses of barbiturates would be more effective than morphine; but I would prefer, in all cases, alkalis.

Since the publication of my paper in your journal, I have received a letter from another doctor stating that a septic condition of the urine had been followed by eclampsia.

Yours, etc.,

WILLIAM JAMES PENFOLD,
Consultant Bacteriologist to the Baker Medical Research Institute, Alfred Hospital, Melbourne.

53, Prospect Hill Road,
Camberwell,
Victoria.

October 4, 1941.

SIR: Dr. Penfold's paper on the nature of eclampsia in your journal of August 23, 1941, was of extraordinary interest to me, as it seemed to throw some light on the effect of adrenaline in the eclamptic. I had used adrenaline (five minims four-hourly) in three severe cases of eclampsia before reading his paper. In the first case, an eclampsia of pregnancy in a patient thought to be actually dying, adrenaline was tried because it was the only thing I could think of that I had not used for her; the improvement (with ultimate recovery) was so dramatic that I have used it for two other severe cases. The only explanation that I could think of at the time for its success was that it might have corrected some "sensitiveness". Perhaps I should add that my eclamptics always have hæmostatic serum (Parke, Davis and Company) and usually "Coramine" or "Anacardone", in addition to sedatives, bowel washouts, and, if possible, alkalis.

Perhaps other practitioners will try out adrenaline in eclampsia, and so determine whether its success in these cases were mere chance or actual fact.

If the diet and dental condition of eclamptics be examined, there will commonly (not always) be found some deficiency in the diet, and evidence of dental sepsis, and not uncommonly a pyelitic history. Indeed in practice I regard eclampsia as a complication of pyelitis. Dr. Penfold is pretty surely right in thinking that an infective element plays its part in causation.

Yours, etc.,

MARY C. DE GARIS.

Geelong,
Victoria.

October 5, 1941.

AUSTRALIA AND POST-WAR IMMIGRATION.

SIR: The leading article in THE MEDICAL JOURNAL OF AUSTRALIA of September 27, 1941, deals with a subject which must interest all if Australia is to survive—that is, post-war migration. That portion which deals with the elimination of physically unfit prospective immigrants needs no comment—such control is essential.

But if there is to be a post-war migration into Australia it is obvious that the immigrants must be obtained not only from Britain, which cannot supply the numbers, but from Europe, and that the Australian attitude to aliens must be altered.

After the last war many British immigrants were attracted to Victoria—some of them by misrepresentation, so they stated. After a short, well-meant course of instruction they were settled on the land at enormous cost. Some of the land was suitable and so were some of the immigrants. But a large number were settled in the north-west Mallee, which is the driest part of Victoria and suffers from periodical droughts. It was a tragedy and was liquidated by keeping the remnant who made good and were given larger areas. I was chairman of a committee provided with some funds by the British Government to look after the others. We managed to place some of them in city or marine occupations, but the balance were repatriated and constituted the worst advertisement Australia ever had.

The remnant, who have done well, were provided by the Bush Nursing Association with a hospital at Werrimull, at which last year there were 37 births and many major and minor operations. This hospital is now closed for want of a doctor. They are isolated and a further disability is inflicted on them.

The majority of people outside Australia regard it as a land of open spaces which could take many people. As a matter of fact, Australia is a comparatively poor country. The most reliable estimates I have seen are that Australia, properly used, could feed 30 million people. The gap between 7 and 30 millions has to be bridged, and that means that Australia must realize her danger and fill the gap.

At present the refugees, except those skilled in medicine, nursing and law, can do any work they like and are mostly doing well. Surely the common sense of the problem must break down prejudices. If immigrants come here they must not be called names like "Pommies", but encouraged to take part in our life. Otherwise the rest of the world will, rightly or wrongly, say we are excluding people but cannot develop the country ourselves.

Of one thing everyone can rest assured. The declining population of Britain offers little hope of filling Australia with British people, and our declining birth rate makes it clear that we cannot do it ourselves. Finally, the birth rate in Australia is much greater in the country than in the cities, and is excellent in tropical Queensland.

Yours, etc.,

JAMES W. BARRETT.

103-105, Collins Street,
Melbourne,

October 6, 1941.

SIR: I would like to make a few comments on your leading article "Australia and Post-War Immigration", of September 27, 1941. I have not had the privilege of reading Dr. F. McCallum's report upon some medical aspects of immigration, but I gather from your article that Dr. McCallum advocates some scheme of post-war immigration and that you have given your approval to such policy.

Let me first consider the general condition of immigration to a country. It is obvious that immigration will occur freely and spontaneously if those who are seeking to immigrate are satisfied that economic, social and/or political conditions are better in the country to which they propose to immigrate than those which appertain to the country in which they live.

In the early days of settlement in Australia people believed that they could and would do better here than in their mother country, and they came here in large numbers, spontaneously, and many brought with them large amounts of capital and they found their belief justified; but it is obvious that for some twenty to thirty years people in the old land have ceased to believe that conditions were better here, and in spite of assisted passages and many inducements the stream of immigrants has practically dried up. The figures in Table I illustrate this. From them you will

TABLE I.
Oversea Migration: Australia.[1]

Period.	Arrivals.	Departures.	Net Migration. (Persons).
1901-05	281,160	297,953	−16,793
1906-10	371,034	313,756	57,278
1911-15	632,820	495,958	136,862
1916-20	539,485	468,776	70,709
1921-25	478,052	294,786	183,266
1926-30	470,480	340,773	129,707
1931-35	239,323	250,209	−10,886
1929	82,248	−70,428	11,820
1930	63,093	71,623	−8,530
1931	40,414	50,508	−10,094
1932	41,997	44,994	−2,997
1933	47,792	47,578	214
1934	53,704	51,424	2,280
1335	55,416	55,705	−289
1936	59,894	58,397	1,497
1937	69,802	64,599	5,203
1938	77,928	68,791	9,137

[1] Net gain in thirty-eight years is 607,934; *minus* sign indicates an excess of departures over arrivals.

see there was a net gain of a little over a quarter of a million in fifteen years (1920 to 1935). In some years there was an actual emigration of some thousands.

I have endeavoured to obtain the cost of this government scheme, but have not found it possible to obtain complete figures, but it cost Australian taxpayers at least £30,000,000. If we reckon land settlement schemes as being part of the immigration plans, the amount of money expended is some £90,000,000. Several schemes have been adopted, and I think I can say all have broken down, even the grandiose plan of the 1925 Development and Migration Commission.

It is thus plain that government planning has failed to attract an appreciable number of immigrants, and it is not likely that any further will succeed. Indeed there are certain factors which would lead one to believe that the chances of success in the future are even more doubtful than the past. One of the main factors in this is that the population of England and Wales is not expanding. Professor Carr-Saunders estimates that it would reach the maximum in 1943, and be under 20,000,000 by 2035, provided the rate of 1933 was maintained; but if the rate of increase falls, as it has been falling, then the population of England and Wales will be 31,000,000 in 1975. If these estimates prove anywhere near correct, then it is not likely that we will get any notable stream of immigrants.

Secondly, I wish now to briefly refer to some observations on the medical aspects of immigration in the past. I have acted as Commonwealth medical referee for the Invalid Pensions Department for some twenty-three years, and I have been struck during the last ten years with the number of immigrants applying for invalid pensions. I cannot give the exact numbers nor the percentages of these applicants to the total immigrants, but I have kept notes of many and I am convinced that great numbers of people were admitted to Australia who were not physically fit and who have become an economic burden. I have examined applicants for invalid pensions (and such is permissible after five years' residence here) who were fifty, sixty and sixty-five years of age when they landed here, and I have seen many cases of pulmonary tuberculosis in young immigrants, cases of mental deficiency, asthma, epilepsy and other disorders, all of which may be grouped under a general term of constitutional inferiority. I could write at much further length upon this subject, but fear to trespass upon your space. My final word is that we should not embark upon any scheme or plan of immigration, but that we should make economic conditions in this land so attractive that people will come here freely.

A prominent Australian economist asserted that one effect (and I may say the only certain effect) of an increase in population is to raise land values, thus benefiting a special class more than the rest of the community fundamentally.

There is only one way to deal with population problems, and that is to make a people happy and prosperous and leave the rest to Nature. In order to do this we must reform our land system, by allowing members of the community to share the benefits of land value.

Yours, etc.,

PAUL G. DANE.

111, Collins Street,
Melbourne,
October 8, 1941.

A NATIONAL MEDICAL SERVICE.

SIR: There is a great deal of robust vigour in Dr. Davey's spirited defence of the existing medical service, but, at the same time, it is uncompromising and painfully stubborn. Without reconnaissance, without parley, he is prepared to defend the citadel and even if necessary make a strategic retreat to the "last ditch".

Is it to be taken for granted that the present system of medical service is the best, that it is adequate and meets every demand, that it can expand and adjust itself according to the increasing interest that the sovereign State takes in the health and welfare of the people? If it can be shown that such is the case let us defend it with all our power and resist vigorously any attempt at change, always, of course, with the interest of the community at heart.

There are many thinking and responsible people who, after careful study, believe, without prejudice, that important and fundamental changes are necessary, because they hold that the system is inadequate and falls short of the demands that are being made on it. It is our duty to confer with these people, to place our views before them and help in any reconstruction if it be found necessary.

The pattern of social life and welfare in which the medical service is incorporated is woven on the frame of basic national finance. It cannot be denied that the weave and even the frame are being rapidly changed, we must see that the threads of medicine are strong and integral in the bold, brave, new pattern. A torn and tattered patch might be the result of stubborn sectional opposition.

Here I repeat from my previous letter "we must be open-minded about the difficulties involved and make an honest attempt to meet them".

Yours, etc.,

JOHN MAUDE.

135, Macquarie Street,
Sydney,
Undated.

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 203, of October 9, 1941.

AUSTRALIAN IMPERIAL FORCE.

Australian Army Medical Corps.

To be Colonels.—Lieutenant-Colonels (Temporary Colonels) H. G. Furnell, D.S.O., J. K. Adey, O.B.E., and F. H. Beare, E.D., 3rd August, 1941.

To be Lieutenant-Colonels.—Major K. C. Purnell, M.C., E.D., Majors (Temporary Lieutenant-Colonels) H. McLorinan, G. B. G. Maitland, D.C.M., G. C. Burston, D. M. Salter and G. G. L. Stening, Majors N. Eadie and J. E. Gillespie, Majors (Temporary Lieutenant-Colonels) R. H. Russell, K. W. Starr and N. H. W. Saxby, 3rd August, 1941.

To be Majors.—Captains R. J. Wheeler and N. H. Morgan, Captains (Temporary Majors) R. S. Smibert, S. L. Seymour and J. S. Peters, and Captain C. H. Johnston, 3rd August, 1941.

AUSTRALIAN MILITARY FORCES.

AUSTRALIAN ARMY MEDICAL CORPS.

Northern Command.

First Military District.

Major J. J. Power, D.S.O., is appointed from the Reserve of Officers (A.A.M.C.), 1st Military District, to command a Casualty Clearing Station, 20th August, 1941.

Honorary Captain I. G. Hooper is appointed from the Reserve of Officers (A.A.M.C.) and to be Captain (provisionally), 25th June, 1940 (in lieu of the notification which appeared in Executive Minute No. 93/1941, promulgated in *Commonwealth Gazette* No. 105 of 1941).

The date of appointment of Honorary Captain J. A. L. Atkinson, which appeared in Executive Minute No. 121/1941, promulgated in *Commonwealth Gazette* No. 136 of 1941, is amended to read "29th May, 1941".

To be Honorary Captain.—Malcolm Wesley Carseldine, 3rd September, 1941.

Eastern Command.

Second Military District.

Captain H. G. B. Bruce is appointed from the Reserve of Officers (A.A.M.C.), 4th September, 1941.

To be Lieutenant-Colonel (temporarily).—Major V. M. Coppleson, 9th August, 1941.

The resignation of Honorary Captain J. B. Cahill of his commission is accepted, 31st August, 1941.

To be Honorary Captain.—Cameron Sutcliffe Rowntree, 4th September, 1941.

Southern Command.

Third Military District.

Honorary Captain G. L. Lillies is appointed from the Reserve of Officers and to be Captain (provisionally), 4th September, 1941.

Captain (Honorary Major) H. Grover is appointed from the Reserve of Officers and to be Major (temporarily) and is seconded, 18th August, 1941.

To be Major (temporarily) and remains seconded.—Captain (provisionally) W. I. E. Mackenzie, 9th September, 1941.

To be Captains (provisionally).—William Henry Hall Cornford, 2nd September, 1941, Edward Ernest Spring and Iver Douglas Macarthur Brodie MacLurkin, 3rd September, 1941, and John Douglas Hicks, 4th September, 1941.

To be Honorary Captains.—Howard Hadfield Eddey, 3rd September, 1941; and Sydney Sunderland and Alan William Richards, 4th September, 1941.

Fourth Military District.

Lieutenant J. Stubbs is appointed from the Reserve of Officers (A.A.M.C.) and to be Lieutenant (Quartermaster), 6th November, 1940.

To be Honorary Captains.—Donald Macdonald Steele, 2nd September, 1941; Elja Haims Levi, Malcolm August Trudinger and Rudolf Herman von der Borch, 4th September, 1941.

Sixth Military District.

To be Honorary Captains.—Randolph Yule Mathew and Kenneth Melville Kelly, 4th September, 1941.

MEDICAL WAR RELIEF FUND.

THE following is a nineteenth list of contributions to the Medical War Relief Fund established by the Federal Council of the British Medical Association in Australia for the relief of distressed medical practitioners in Great Britain.

Queensland.

£5 5s.: Dr. R. M. Murray, Dr. R. Fulton Craig.
£2 2s.: Dr. A. S. Furness.
£1 1s.: Dr. Nettie G. Hill.

Tasmania.

£50: Dr. W. K. McIntyre.
£5: Major J. Bean (second contribution).
£2 2s.: Dr. F. C. Klausen.
£1 11s. 6d.: Dr. T. J. Ick.
£1 1s.: Dr. T. Goddard.

Nominations and Elections.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Selle, Hal, M.B., B.S., 1939 (Univ. Sydney), NX436, Captain, A.A.M.C., R.M.O. 2/31 Australian Infantry Battalion, Australian Imperial Force, Abroad.

The undermentioned have been elected members of the New South Wales Branch of the British Medical Association:

Duncan, Ian Lovell, M.B., B.S., 1940 (Univ. Sydney), NX35135, Captain, A.A.M.C., Headquarters, Royal Australian Engineers, 8th Australian Division, Abroad.
Finlay, Donald Murray Logier, M.B., 1940 (Univ. Sydney), c.o. Naval Liaison Officer, Australia House, London.
Finlay, Sinclair Elwyn, M.B., 1940 (Univ. Sydney), c.o. Naval Liaison Officer, Australia House, London.
Barnes, Winstanley, M.B., B.S., 1935 (Univ. Sydney), 10, Victoria Street, Mayfield.
Windeyer, John Spencer, M.B., B.S., 1941 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Willson, John Hector, M.B., B.S., 1939 (Univ. Sydney), 8, Birriga Road, Bellevue Hill.
Sullivan, John Francis, M.B., B.S., 1939 (Univ. Sydney), NX12279, Captain, A.A.M.C., R.M.O. 2/17 Battalion, Australian Imperial Force, Abroad.
Marshall, Charles Edward, M.B., 1940 (Univ. Sydney), 12, Fairfax Road, Bellevue Hill.
Kent, Merrian Winifred, M.B., B.S., 1941 (Univ. Sydney), 6, Hiawatha Flats, 63, Douglas Street, Stanmore.
Hodgson, George Alfred, M.B., B.S., 1939 (Univ. Sydney), 135, Denison Road, Dulwich Hill.
Blacket, Ralph Beattie, M.B., B.S., 1941 (Univ. Sydney), 105, The Avenue, Hurstville.

Obituary.

EDWARD HERRICK KNOWLES.

WE regret to announce the death of Dr. Edward Herrick Knowles, which occurred on October 19, 1941, at Sydney.

ALFRED WYNTER HAWTHORNE.

WE regret to announce the death of Dr. Alfred Wynter Hawthorne, which occurred on October 19, 1941, at Carcoar, New South Wales.

Notice.

THE New South Wales Post-Graduate Committee in Medicine announces that the next clinico-pathological conference arranged by the Post-Graduate Directors of Medicine, Surgery and Pathology, will be held in the lecture hall at the Prince Henry Hospital, Little Bay, New South Wales, on Monday, October 27, 1941, at 4.30 p.m. The subjects will be cases for diagnosis and discussion, and a cordial invitation to be present at the meeting is extended to all medical practitioners.

THE New South Wales Police Department announces the theft of a quantity of morphine powder, "M & B 693" tablets, tablets of drugs of the sulphanilamide group and miscellaneous medical supplies. Medical practitioners to whom drugs of this kind are offered for sale in suspicious circumstances are asked to notify the Police Department, Sydney, telephone B030, extension 231, or the nearest police station.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such a notification is received within one month.

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rates are £2 for Australia and £2 5s. abroad *per annum* payable in advance.